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.

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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

establishing the diagnosis. However, the final diagnosis relied on pathological investigations.

In spite of the benign nature of myxomas, they may inevitably cause valvular dysfunction and secondary pulmonary hypertension and have a significant propensity to embolize the pulmonary artery (1). Pulmonary artery and pulmonary valve myxomas have the common features of right heart system, such as predilections of right ventricular obstruction, right-sided valve insufficiencies, and pulmonary embolism; however, their special characteristic is a smaller size. Pulmonary myxomas could occur isolated or in association with other congenital heart defects or acquired disorders. There were more myxomas arising from the pulmonary valve than from the pulmonary artery or from pulmonary valve and pulmonary artery. Most pulmonary valve myxomas arose from the valve leaflets, and most pulmonary artery myxomas arose from the main pulmonary artery. Because of potential hazards and occasional misdiagnosis, the patients endow an early surgical treatment upon diagnosis (5). Most patients warranted a surgical resection of the myxoma under standard cardiopulmonary bypass, while some patients were operated under normothermic cardiopulmonary bypass or deep hypothermic circulatory arrest. Concurrent procedures to myxoma resection, such as pulmonary valve repair or replacement, or right ventricular outflow tract reconstruction should be performed simultaneously. An early surgical treatment is warranted upon diagnosis because of potential hemodynamic disturbances and predilection of embolization. Most patients have a good prognosis following surgical treatment.

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Defibrillator lead dislocation after manual lumbar traction

To the Editor,

We report a 63-year-old patient with prior coronary artery bypass surgery and recent history of recurrent hospital admissions for refractory heart failure because of ischemic cardiomyopathy and sustained ventricular tachycardia. The patient underwent ICD implantation through left subclavian vein approach (Medtronic, single chamber, model-Maximo II VR, D284VRC, with 6947 ventricular active fixation lead). This case report describes the first patient, to our knowledge, with defibrillator lead dislocation after manual lumbar traction for low back pain. The patient was admitted to the emergency service with severe chest pain, and electrocardiography revealed 0.5–1 mm ST-segment elevation in leads DII, DIII, and aVF. After initial evaluation, coronary angiography and percutaneous coronary intervention were immediately performed with the diagnosis of acute inferior myocardial infarction. Case history revealed ICD implantation 4 months ago because of ischemic cardiomyopathy and sustained ventricular tachycardia. During angiography, abnormal course of the defibrillator lead was noticed (Fig. 1). ICD interrogations revealed a dislocated defibrillator lead with lead impedance over 2.000 ohms and inability to capture, and defibrillator analysis showed no ventricular sensing and pacing. Despite successful primary percutaneous coronary intervention for totally occluded circumflex coronary artery, the patient developed shock and expired the day after. When relatives were questioned, it was learned that the patient had undergone manual lumbar traction by a non-medical person because of low back pain.

Lumbar traction has been used since prehistoric times for spinal disorders. The most commonly used traction technique is manual traction exerted by non-medical persons, using the patient's body weight to apply force. Manual traction is applied as the non-medical person's hands and/or belt are used to pull the patient's legs (1). Traditional lumbar traction force was applied to the thorax in the cephalad direction and to the pelvis and ankles in the caudal direction with the subjects positioned supine (1). Generally, pelvic belt with straps are used for distraction. In our country, non-medical persons commonly use manual lumbar traction as an alternative treatment for low back pain.

Literature search did not reveal any case of pacemaker lead dislodgement after manual lumbar traction. However, there is a case report showing isolated ureter injury after traction for the low back pain (2).

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