A 56-year-old hypertensive and diabetic male, with previous triple coronary artery by-pass grafts, was admitted to our hospital for non-ST elevation MI. Angiography revealed patency of left internal mammary artery to left anterior descending artery (LAD), occlusion of saphenous vein grafts to right coronary artery (RCA) and circumflex (LCx), chronic total occlusion (CTO) of RCA and LAD and severe lesion of distal left main (LM) involving the bifurcation between intermediate artery (IA) and LCx (Medina 1, 0, 0) (Fig. 1).

Percutaneous coronary intervention of protected LM was decided. After advancing a guidewire to distal IA (Fig. 2A), the lesion was pre-dilated with a 3.0 × 12-mm balloon at 14 atm (Fig. 2B). An ABSORB bioresorbable vascular scaffold BVS 3.5 × 18-mm (Abbott Vascular, Santa Clara, CA) was therefore implanted at 14 atm with an expected diameter of 3.94-mm (Fig. 2C). A final good angiographic result was eventually present with preserved flow in IA and LCx (Fig. 2D). Patient was discharged asymptomatic and 30-day follow-up was uneventful.

Two major points about ABSORB BVS use could be drawn from our report. First of all, it is known that some concerns have been previously raised about implantation of an ABSORB BVS in a coronary artery with a diameter higher than 3.5-mm [1]. In the ABSORB Cohort B study a single size scaffold (3.0 × 18-mm) was available, limiting therefore the implantation to vessels with an estimated diameter of 2.5 to 3.3-mm. Data about ABSORB BVS implanted in a coronary artery larger than 3.3-mm have shown higher incidence of malapposition or fracture, [2–4]. Our case report shows for the first time the feasibility of ABSORB BVS implantation in a coronary artery with an approximated diameter of 4-mm, using a 3.5-mm device, which can theoretically be implanted at 16 atm with an expected diameter of 4.01-mm.

Another concern for left main treatment by ABSORB BVS is the presence of bifurcation. Although in our case LAD was ostially occluded, a Medina 1, 0, 0 bifurcation was present between IA and LCx. Bifurcations currently represent an exclusion criteria for ABSORB BVS implantation, due to the possible fracture of the device in case a post dilatation towards the side branch or a kissing balloon is needed [5,6]. With this regards, our case demonstrates the feasibility of ABSORB BVS implantation in bifurcation lesions in case no lesion is present at the ostium of the side branch in order to avoid any rewiring of the scaffold and post-implantation maneuvers, which can damage the scaffold.

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Fig. 1. Coronary angiography shows distal LM lesion (80%), CTO of ostial LAD, diffuse atheromatosis without ostial involvement of IA (main vessel) and LCx (side branch), forming a bifurcation lesion (Medina 1, 0, 0); and absence of significant lesions in IA and LCx. Pre-procedural reference vessel disease of LM: 3.82-mm.

Fig. 2. A: Advance of a BMW guidewire (0.014×190) (Abbott Vascular, Santa Clara, CA) to distal IA through a 6F EBU 3.5 guide catheter. B: Pre-dilatation of distal LM and IA with a 3.0×12-mm TREK balloon at 14 atm (Abbott Vascular; Santa Clara, CA). C: Implantation of bioresorbable vascular scaffold BVS 3.5×18-mm at 14 atm (expected diameter: 3.94-mm). D: Final result with adequate stent expansion, absence of plaque shifting and preserved flow in IA and LCx.

References

The impact of G5665T polymorphism of endothelin-1 gene, on endothelin-1 levels and left ventricular function in ischemic heart disease

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It is well established that heart failure (HF) represents a complex clinical syndrome with several factors contributing to its pathogenesis and although the pathophysiology is closely related to the etiology, there are still aspects requiring further clarification. Data have shown that endothelin-1 (ET-1) levels, known for its vasoconstrictory activity, are increased in states of HF [12], while these have been correlated with structural and functional changes in the myocardium of patients with HF [3], with the severity of HF [4], while there is evidence that ET-1 may have a role in the prognosis of patients with HF [5,6]. Moreover, several genetic polymorphisms have been proposed to affect ET-1 levels and subsequently the pathophysiology of heart failure including functional and structural aspects as well as the risk of HF [7,8]. Previous studies have shown that a well known polymorphism the G5665T on ET-1 gene has been associated with increased risk of hypertension [9,10]. However, there is no clear evidence for a role of this polymorphism in patients exhibiting HF. Therefore in the present study we examined the effect of G5665T polymorphism on endothelin-1 gene, on the left ventricular (LV) function and ET-1 levels.

The study population consisted of 198 patients (mean age 70.1± 0.68) with ischemic heart disease with stable mild to moderate chronic HF (New York Heart Association functional classes II–III) and left ventricular ejection fraction <40% on echocardiography, and 160 healthy controls (mean age 60.1 ± 0.73). The etiology of HF was ischemic based on coronary angiographic examination (coronary artery disease was defined as at least 1-vessel disease with >75% narrowing of the luminal diameter), echocardiography, exercise test and disease history. Hypercholesterolemia was defined as fasting cholesterol levels >200 mg/dl or use of lipid-lowering agents during the past 6 months. Hypertension was defined as a systolic blood pressure >140 mm Hg, a diastolic blood pressure >90 mm Hg, or current use of antihypertensive medication. Diabetes mellitus was defined as fasting glucose >110 mg/dl, non-fasting glucose >160 mg/dl or a history of treatment of diabetes. Exclusion criteria were any acute or chronic inflammatory disease, malignancies, recent acute coronary event during the last 8 months, and renal or liver failure. The study was approved by the Institutional Ethics Committees, and an informed consent was given by all the participants.

Venous blood samples were obtained from all patients during admission to our department. After centrifugation at 3500 rpm at 4 °C for 15 min, plasma or serum was collected and stored at 80 °C until assayed. Enzyme linked immunosorbent assay was used to determine plasma levels of endothelin-1 (Biomedica Inc., Austria). Genomic DNA was extracted from 2 to 5 ml of whole blood using standard methods (QIAamp DNA blood kit; Qiagen, West Sussex, UK). For the detection of G5665T (rs5370) polymorphism on the ET-1 gene, we used primer pairs to amplify a part of the gene by Polymerase Chain Reaction (PCR) with the following flanking intronic primers: rs5370F: 5′-TCTTGCTTTTATAGGTCGGAGCC-3′ and rs5370R: 5′-TTTGAAGGAGGCTGTC-3′. The resulting product (262 bp) (95 °C for 10 min, 35 cycles × [95 °C for 1 min, 61 °C for 1 min, 72 °C for 1 min and 30 s, 72 °C for 10 min], hold at 4 °C) was digested by Cac8I restriction endonuclease (16 hour incubation at 37 °C and resolution by electrophoresis at 2% agarose gel). Digested fragments were visualized after ethidium bromide staining under ultraviolet light. For PCR quality control, 10% of the samples was randomly selected and genotyped twice for quality assurance, which yielded 100% concordance. The SPSS version 18.0 (SPSS, PASW, Chicago, IL) software was used for all the statistical calculations.

A significant difference was found in serum lipids, prevalence of hypertension, and diabetes mellitus between the control group and patients with HF (p < 0.05). Moreover, we found that the ET-1 levels were higher in the IHF group compared to those in the control group (2.21±0.11 vs 1.13±0.10, p<0.0001). Importantly, we observed that the G5665T polymorphism on ET-1 gene affects significantly the ET-1 levels. Although no significant difference was observed in the control group between TT homozygotes and G allele carriers (1.51±0.50 vs 1.20±0.10, p = NS), we observed a significant difference in the IHF group.

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