Left ventricular (LV) dysfunction represents an important clinical predictor of stent thrombosis after bare-metal stent (BMS) and drug-eluting stent (DES) implantation. Reduced left ventricular (LV) ejection fraction (EF) is considered a risk factor for this complication after both bare-metal stent (BMS) and DES implantation. Therefore, the aim of this study was to evaluate the safety of DES on long-term follow-up in patients with LV dysfunction undergoing percutaneous coronary intervention. We retrospectively selected all patients with an EF <45% undergoing percutaneous coronary intervention with implantation of ≥1 sirolimus- or paclitaxel-eluting stent at our institution. The primary endpoint of the study was all-cause mortality, retrieved using both Social Security Database and hospital records. We also compared the results of this group with a historical cohort of patients with LV dysfunction undergoing BMS implantation; 121 patients who received ≥1 DES were enrolled. The mean LVEF was 36 ± 8%, with 20 patients (16%) with a LVEF ≤25%; 36 patients (30%) had diabetes mellitus, and DES implantation was considered off-label in 100 patients (83%). Survival at 1-, 2-, and 3-year follow-up was 94% (95% confidence interval [CI] 88 to 100), 90% (95% CI 82 to 98) and 88% (95% CI 80 to 96), respectively. In conclusion, the favorable results of this study demonstrate the safety of DES in patients with LV dysfunction. © 2008 Elsevier Inc. All rights reserved. (Am J Cardiol 2008;102:679–682)
The primary endpoint of the study was all-cause mortality. The follow-up data were retrieved using both the Social Security Database and hospital records. We also compared the results of this group of patients with a historical cohort of patients who underwent BMS implantation before the introduction of DES between May 1996 and March 1999.

Non-Q-wave myocardial infarction (defined as an elevation of creatinine kinase-MB \( \geq 3 \) times the upper limit of normal), urgent coronary revascularization (urgent bypass surgery or urgent PCI). Prospective data was collected for each patient from the time of PCI for a follow-up period ranging 18 to 51 months (median 30 months).

The clinical and procedural characteristics of the historical cohort of patients who underwent BMS implantation were compared with those of the DES cohort. The follow-up data were retrieved using both the Social Security Database and hospital records. We also compared the results of this group of patients with a historical cohort of patients who underwent BMS implantation before the introduction of DES between May 1996 and March 1999.

Statistical analysis was performed with SPSS 11.0 software for Windows (SPSS, Inc., Chicago, Illinois). Continuous variables were reported as mean ± SD; discrete variables are presented as percentages. The Student’s t test was used to compare continuous variables, and the chi-square test (or the Fisher’s exact test when indicated) was performed to compare discrete variables between the 2 groups. Kaplan-Meyer survival curves were used to compare time-dependent variables, using the Cox regression analysis for multivariable analysis. Survival rates and 95% confidence intervals (CI) are presented; 2-tailed p values of 0.05 were considered statistically significant for this analysis.
tion were similar to the cohort of patients who underwent DES implantation. In particular, the mean calculated LVEF was 39 ± 10%, and 59% of patients had multivessel coronary artery disease; of these, 18% had multivessel PCI. A lower percentage of patients received β blockers and statins at the time of the procedure compared with the DES group (data not shown), and a prophylactic intra-aortic balloon pump was inserted in 10 patients (9%) in the BMS group. For the DES cohort, median duration of follow-up was 30 months (range 18 to 51), and there was no in-hospital death; 2 patients (2%) had myocardial infarction, and 1 of these (1%) required urgent repeat PCI for in-stent thrombosis. No patients underwent urgent coronary artery bypass surgery.

Actuarial survival curves were created showing a 1-, 2-, and 3-year survival of 94% (95% CI 88 to 100), 90% (95% CI 82 to 98), and 88% (95% CI 80 to 96), respectively. Long-term survival appeared similar in patients with LVEF ≤25% and those with LVEF 26 to 45% (5% vs 13%, p = 0.45). Figure 1 shows survival curves for the cohort of patients with DES and, for comparison, of patients with BMS.

Discussion

The present study is reassuring because it shows that despite concerns regarding an increased incidence of late stent thrombosis in patients with depressed LVEF, the use of DES appears safe and is associated with a favorable long-term outcome.

DES significantly reduce the incidence of angiographic restenosis and subsequent target vessel revascularization across a large spectrum of lesions and patients subsets. Initial studies had suggested that rates of mortality and myocardial infarction are not reduced by DES in clinical randomized trials and observational registries, especially if DES were implanted for “off-label” indications, and concerns have been raised that the lack of effects of DES on “hard” clinical end points may be explained by an increased incidence of stent thrombosis, especially late stent thrombosis. Although more recent data suggest that the increase in late stent thrombosis may be less than initially described and may be offset by a beneficial impact on late ischemic events, we considered that depressed LVEF has been shown to be an independent predictor of subacute stent thrombosis possibly due to “slow flow” within the stent and, therefore, LV dysfunction and DES could have represented a risky combination. Thus, we decided to retrospectively review the cohort of patients who had received a DES. Our results are in line with the recent reports.

Although speculative, prevention of in-stent restenosis observed with DES may lead to improvement of long-term survival, balancing potential adverse events related to stent thrombosis, especially in patients with LV dysfunction. In fact, contrary to traditional belief, in-stent restenosis is not a “benign clinical entity.” Numerous studies demonstrated that 30% to 60% of cases of BMS restenosis presented as acute coronary syndromes. The arterial injury after stent implantation and resulting neointimal proliferation may serve as a substrate for subsequent thrombus formation, leading to an acute presentation. Bossi et al and the Prevention of Restenosis with Tranilast and its Outcomes (PRESTO) investigators also observed a poor outcome in patients with unstable angina pectoris or myocardial infarction related to angiographic evidence of in-stent restenosis. Interestingly, LV dysfunction has been associated with an increased risk of acute presentation of stent restenosis.

Several limitations of this study, such as the retrospective nature of the analysis and the relatively small number of patients, need to be acknowledged. Also the historical BMS cohort is not an ideal control for the current DES cohort, and comparisons should be interpreted cautiously. Moreover, we were not able to collect more specific data from long-term follow-up, such as incidence of myocardial infarction, repeat revascularization, and in-stent thrombosis, therefore limiting the analysis to the assessment of mortality only.


