Randomized Comparison of GR-II Stent and Palmaz-Schatz Stent for Elective Treatment of Coronary Stenoses

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Background—This prospective multicenter randomized clinical trial was designed to evaluate the long-term angiographic and clinical outcomes of elective treatment with the GR-II stent compared with the Palmaz-Schatz (PS) stent in patients with coronary stenoses.

Methods and Results—Seven hundred fifty-five patients with myocardial ischemia and de novo native coronary stenoses in 3- to 4-mm vessels were randomly assigned to the PS (375 patients) or the GR-II stent (380 patients). The primary end point was 12-month target lesion revascularization (TLR)-free survival. Angiography was performed at baseline and at follow-up in the first 300 consecutive patients to assess the frequency of angiographic restenosis. Procedure success was 98.5% for the GR-II stent and 99.4% for the PS stent (P<0.01). At 30 days, patients assigned to the GR-II stent had a higher stent thrombosis rate (3.9% versus 0.3% for PS stent, P<0.001) and TLR rate (3.9% versus 0.5% for PS stent, P<0.001). The GR-II group had a higher follow-up restenosis frequency (47.3% versus 20.6% for the PS group, P<0.001) and a lower 12-month TLR-free survival rate (71.7% versus 83.9% for the PS group, P<0.001). Multivariate logistic regression analysis identified a smaller final stent minimal lumen diameter (odds ratio [OR] 2.49, 95% CI 1.56 to 3.98; P<0.001), diabetes mellitus (OR 2.14, 95% CI 1.42 to 3.22; P<0.001), and use of the GR-II stent (OR 1.78, 95% CI 1.20 to 2.64; P<0.001) as independent determinants of 12-month TLR.

Conclusions—On the basis of these long-term follow-up data, we conclude that use of the GR-II stent should be limited to the acute treatment of abrupt or threatened closure after failed conventional balloon angioplasty procedures.

Key Words: angioplasty stents restenosis
Methods

Patient Population

Patients providing informed consent were included in the study if they had objective evidence of myocardial ischemia, a de novo native coronary lesion in a vessel between 3.0 to 4.0 mm in diameter by visual estimate, and required 1 or two 20-mm or a single 40-mm GR-II stent or 1 or two 15-mm PS stents. Lesions up to 30 mm in length were included. Patients were excluded if there was evidence of a recent (<7-day) myocardial infarction, angiographic thrombus, left ventricular ejection fraction <35%, unprotected left main disease, or unplanned stent use, such as the occurrence of a significant dissection or reduced (Thrombolysis in Myocardial Infarction [TIMI] grade 2) flow after conventional PTCA.

Adjunct Pharmacology

The protocol recommended that all patients be premedicated with aspirin (325 mg daily) and ticlopidine (250 mg twice daily) for 48 hours before the procedure. Bolus intravenous heparin was administered to maintain the activated clotting time between 250 to 350 seconds. After the stent procedure, all patients received aspirin (325 mg) indefinitely and ticlopidine (250 mg twice daily) for 4 weeks. Postprocedural administration of unfractionated heparin, low molecular weight heparin, coumadin, or antiplatelet agents were left to the discretion of the operator.

Stent Deployment Method

Predilation using conventional PTCA techniques was performed before stent implantation in all patients. For patients assigned to the PS stent, a 3.0-, 3.5-, or 4.0-mm PS stent was deployed at 6 to 8 atm with use of the stent delivery balloon to approximate a 1.1/1.0 stent-to-artery ratio. After dilatation, high-pressure (16- to 18-atm) inflation was recommended; a noncompliant PTCA balloon was used in an attempt to achieve a <10% final diameter stenosis. In patients assigned to the GR-II stent, the protocol recommended that a slightly oversized GR-II stent (0.1 to 0.5 mm larger than the reference) be selected to achieve a 1.1–1.2 to 1.0 stent–to–reference artery ratio. Only GR-II stents between 3.0, 3.5, and 4.0 mm in diameter were available for this study. The GR-II stent was deployed at 6 to 8 atm with use of the stent delivery balloon; subsequent high-pressure (14- to 16-atm) inflations were performed with the use of a semicompliant or noncompliant balloon.

Data Collection and End Points

Detailed case report forms were completed by the clinical coordinator at each site and were forwarded to the data analysis center (MED Institute, West Lafayette, Ind) after independent monitoring. Clinical follow-up was obtained at 30 days and 12 months. All adverse clinical events were adjudicated by an independent clinical events committee (CDAC, Department of Medicine, Beth Israel Hospital, Boston, Mass). All procedural and follow-up cineangiograms were obtained by using standard acquisition guidelines and were submitted to an independent Angiographic Core Laboratory (Washington Hospital Center, Washington, DC). Follow-up cineangiograms were obtained in the first 300 consecutive patients at >9 months. Quantitative angiographic analysis was performed by use of the CMS-GFT system (Medis). Standard morphological criteria were used to characterize baseline lesion complexity and to identify the occurrence of angiographic complications.

The prespecified primary end point of the trial was the 12-month target lesion revascularization (TLR)-free survival. Secondary end points included procedure success, defined as the attainment of <50% diameter stenosis in the absence of death or emergent bypass surgery, stent thrombosis, and major adverse cardiac events, including death, myocardial infarction, and TLR at 30 days and 12 months. Myocardial infarction was defined as a rise in total creatinine kinase >2 times normal with a positive MB fraction in association with any of the following criteria: (1) ischemic chest pain lasting >30 minutes, (2) a new 2-step Minnesota Code Q wave, (3) persistent ST- or T-wave changes, or (4) a new left bundle branch block. Stent thrombosis was defined as an angiographically or clinically documented coronary occlusion (TIMI grade 0 flow) at the stent site or cardiac death within 30 days of the procedure. The secondary angiographic end point was the restenosis frequency, defined as a >50% follow-up diameter stenosis within the stent. The mean reference diameter (RD) and the minimal lumen diameter (MLD) within the axial stent length were used to calculate the percent diameter stenosis, defined as (1 − MLD/RD)×100, within the stent. MLD 0.0 was imputed in the presence of a total occlusion at baseline or follow-up. Acute gain (in millimeters) was defined as the change in the stent MLD from baseline to the final procedural angiogram; late loss (in millimeters) was defined as the change in stent MLD from the final to the follow-up angiogram. The regression loss index within the stent was defined as the regression coefficient of the relationship between late loss (y-axis) and acute gain (x-axis). Stent recoil, defined as (final balloon MLD−final stent MLD)/RD, was measured as an index of the maximal loss in lumen dimensions resulting from the final balloon deflation.

Statistical Analysis

The sample size determination for this trial was based on the equivalency methods of Blackwelder and Chang. The null hypothesis for the study was that 12-month TLR-free survival rate was at least 8% lower or higher for patients treated with the GR-II stent than for those treated with the PS stent. The alternate hypothesis was that the difference in 12-month TLR-free survival rates between the 2 groups was <8%. With an estimated 85% 12-month TLR-free survival rate in PS stent–treated patients, 341 patients (682 total patients) would be required per arm to achieve a statistical power of 90% with a type I (α) error of 0.05. Assuming a 10% dropout rate due to patient loss to clinical follow-up, 750 patients were required for the present study.

Patient outcomes were analyzed by using an “intention-to-treat” analysis. Continuous variables are reported as mean±SD, and all binary and ordinal variables are presented as frequencies. Comparisons between patients treated with the GR-II stent and the PS stent were performed by use of t tests or Wilcoxon nonparametric tests for continuous variables; ordinal variables were compared by χ² analysis or the Fisher exact test. Multivariable logistic analyses were performed to identify clinical and angiographic predictors of (1) 30-day stent thrombosis, (2) 12-month TLR, and (3) angiographic restenosis. Variables included in the model were reference vessel size, location of the left anterior descending coronary artery, lesion length, final MLD, stent length–to–lesion length ratio, total length of implanted stent, stent–to–artery ratio, GR-II stent use, diabetes mellitus, and “incorrect” GR-II stent sizing (undersized and oversized). Binary stepwise multivariate logistic regression analysis used a value of P<0.10 for entry and P<0.20 for removal. A 2-tailed value of P<0.05 was considered significant.

Results

Procedural Findings

Between January and October 1996, 755 patients at 31 clinical sites (Appendix) were randomly assigned to elective stent placement with the GR-II stent (380 patients) or the PS stent (375 patients). Six patients in the GR-II stent group and 7 in the PS stent group had failed stent deployment and did not receive a stent or received an alternate stent. Baseline
Early Clinical Outcomes

At 30 days, composite major adverse cardiac events were significantly higher in the GR-II stent group ($P=0.029$) because of a higher need for 30-day TLR ($P<0.01$) and a higher incidence of stent thrombosis ($P<0.001$) (Table 3). By multivariate logistic regression analysis, a low stent-to-artery ratio (odds ratio [OR] 0.006, 95% CI 0.00 to 0.14; $P<0.01$), use of the GR-II stent (OR 13.56, 95% CI 1.58 to 116.06; $P=0.02$), and the presence of diabetes (OR 3.13, 95% CI 1.01 to 9.65; $P<0.05$) were independent determinants of 30-day stent thrombosis. In addition, incorrect GR-II stent sizing was a univariate predictor of stent thrombosis (OR 8.22, $P=0.04$). Mortality was low in both the GR-II (0.3%) and the PS (0.5%) stent groups.

Late Clinical Outcomes

Complete 12-month clinical follow-up was available in 97.1% of the GR-II patients (6 patients were lost to follow-up, and 5 patients were not treated) and in 97.6% of the PS patients (6 patients were lost to follow-up, and 3 patients were not treated) (Table 3). The primary end point of the study, 12-month TLR-free survival rate, was significantly lower for the GR-II stent group than for the PS stent group (71.7% versus 83.9%, respectively; $P<0.001$) (Figure 1). Multivariate logistic regression analysis identified a smaller final stent MLD (OR 2.49, 95% CI 1.56 to 3.98; $P<0.001$), diabetes mellitus (OR 2.14, 95% CI 1.42 to 3.22; $P<0.001$), and use of the GR-II stent (OR 1.78, 95% CI 1.20 to 2.64; $P<0.01$) as independent determinants of TLR. Major adverse cardiac events at 12 months were significantly higher in the GR-II arm because of a greater need for TLR ($P<0.001$). TLR rates tended to be higher for patients with oversized (27.9%) and undersized (26.0%) compared with correctly sized (24.2%) GR-II stent selection, and site-to-site variability in 12-month TLR was substantially greater in the GR-II-treated patients.

Follow-Up Angiographic Findings

Follow-up angiography was available in 217 of the 306 prespecified patients (72% of GR-II and 70% of PS stent...
restenosis rate was significantly higher in GR-II–treated patients. The likely explanation for increased early complications associated with the GR-II stent in this clinical trial is the reduced stent-to-artery ratio resulting from (1) increased acute recoil of this coiled stent design and (2) implantation of undersized stents (an operator-dependent variable). Other studies have demonstrated that small stent size and incompletely expanded stents are important predictors of subacute stent thrombosis.9

There are a number of potential (and perhaps interrelated) reasons for the disappointing late outcomes of the GR-II stent compared with the PS stent. First, late patient outcomes (both angiographic and clinical restenosis rates) were a smaller final MLD, diabetes mellitus, and implantation of a GR-II stent. The likely explanation for increased early complications associated with the GR-II stent in this clinical trial is the reduced stent-to-artery ratio resulting from (1) increased acute recoil of this coiled stent design and (2) implantation of undersized stents (an operator-dependent variable). Other studies have demonstrated that small stent size and incompletely expanded stents are important predictors of subacute stent thrombosis.9

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TABLE 3. Clinical Outcome

<table>
<thead>
<tr>
<th>In-hospital events, n (%)</th>
<th>GR-II Stent (n=380)</th>
<th>PS Stent (n=375)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>MACE (death, Q-MI, TLR)</td>
<td>12 (3.2)</td>
<td>3 (0.8)</td>
<td>0.04</td>
</tr>
<tr>
<td>Death</td>
<td>1 (0.3)</td>
<td>2 (0.5)</td>
<td>0.62</td>
</tr>
<tr>
<td>Q-MI</td>
<td>5 (1.3)</td>
<td>2 (0.5)</td>
<td>0.45</td>
</tr>
<tr>
<td>TLR (PTCA or CABG)</td>
<td>10 (2.6)</td>
<td>0 (0.0)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Re-PTCA</td>
<td>8 (2.1)</td>
<td>0 (0.0)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>CABG</td>
<td>4 (1.1)</td>
<td>0 (0.0)</td>
<td>0.12</td>
</tr>
<tr>
<td>30-d cumulative events, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MACE (death, Q-MI, TLR)</td>
<td>16 (4.2)</td>
<td>5 (1.3)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Death</td>
<td>1 (0.3)</td>
<td>2 (0.5)</td>
<td>0.62</td>
</tr>
<tr>
<td>Q-MI</td>
<td>5 (1.3)</td>
<td>2 (0.5)</td>
<td>0.45</td>
</tr>
<tr>
<td>TLR (PTCA or CABG)</td>
<td>15 (3.9)</td>
<td>2 (0.5)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Re-PTCA</td>
<td>10 (2.6)</td>
<td>0 (0.0)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>CABG</td>
<td>7 (1.9)</td>
<td>1 (0.3)</td>
<td>0.08</td>
</tr>
<tr>
<td>Stent thrombosis</td>
<td>15 (3.9)</td>
<td>1 (0.3)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

12-mo follow-up,* n (%) |                  |                 |   |
| MACE (death, Q-MI, TLR) | 110 (29.8)       | 65 (17.8)       | <0.001 |
| Death                   | 10 (27)           | 10 (2.7)        | 0.98 |
| Q-MI                    | 5 (1.4)           | 3 (0.8)         | 0.73 |
| Non-Q-MI                | 50 (13.6)         | 41 (11.2)       | 0.41 |
| TLR (PTCA or CABG)     | 101 (27.4)        | 56 (15.3)       | <0.001 |
| CABG                   | 19 (5.1)          | 16 (4.4)        | 0.76 |
| PTCA                   | 84 (22.8)         | 40 (10.9)       | <0.001 |

MACE indicates major adverse cardiac events; Q-MI, Q-wave myocardial infarction.

*At 12-mo follow-up, there were 369 GR-II stent patients and 366 PS stent patients.

Figure 1. TLR-free survival: Kaplan-Meier estimate of overall survival free of TLR. Freedom from TLR at 12 months was 71.7% for GR-II stent vs 83.9% for PS stent (P<0.001).

Figure 2. Cumulative distribution curves for MLD at baseline, final procedure, and 9-month follow-up in patients randomized to GR-II and PS stent. MLDs at baseline are similar in both groups; however, final procedure MLDs (2.64±0.41 mm for GR-II stent vs 2.83±0.43 mm for PS stent, P<0.001) and follow-up MLDs (1.48±0.73 mm for GR-II stent vs 1.90±0.74 mm for PS stent, P<0.001) were significantly smaller for GR-II–treated patients.

5.05; P=0.014), and longer lesion length (OR 1.06, 95% CI 1.02 to 1.10; P=0.003) as independent determinants of restenosis. Total stent length was a univariate (OR 10.43, P<0.001) but not a multivariate predictor of restenosis.

Discussion

In the present study comparing elective stent treatment of native coronary stenoses, patients treated with the GR-II stent had more early complications (stent thrombosis and 30-day composite events) and higher late angiographic and clinical restenosis rates than did the patients treated with the PS stent. Independent predictors of 12-month TLR-free survival (the primary end point of this randomized trial) were a smaller final MLD, diabetes mellitus, and implantation of a GR-II stent.

The likely explanation for increased early complications associated with the GR-II stent in this clinical trial is the reduced stent-to-artery ratio resulting from (1) increased acute recoil of this coiled stent design and (2) implantation of undersized stents (an operator-dependent variable). Other studies have demonstrated that small stent size and incompletely expanded stents are important predictors of subacute stent thrombosis.9
present study inadvertently resulted in a much higher total stent length and stent length-to-lesion length ratio for the GR-II stent (29.3 versus 22.0 mm for the PS stent \( P<0.001 \) and 2.5 versus 1.5 for the PS stent \( P<0.001 \), respectively). In this trial and others, stent length has been an important predictor of late angiographic and clinical restenosis. Third, as with any new device, operator technique is critical for achieving optimal results.11 There was a marked site-to-site variability in long-term patient outcomes in the GR-II group, a finding not seen in the PS stent group. It is possible that a different stent implantation technique (ie, a larger balloon-to-artery ratio to offset the acute recoil) and tissue prolapse, resulting in lower final procedural diameter stenoses) would have produced better results in the GR-II arm of the present study. In 2 other randomized clinical trials (albeit in patients with acute myocardial infarction) comparing the GR-II stent with balloon angioplasty (the GR II Stent in Acute Myocardial Infarction [GRAMI] and the florence randomized elective stenting in Acute coronary occlusion [FRESCO] trials), the long-term patient outcomes in the stent groups were significantly better than the long-term outcomes in the present study.12,13 The present study cannot address the mechanism of increased late lumen narrowing in patients treated with GR-II stents. Serial intravascular ultrasound studies have shown that late stent recoil is minimal with tubular slotted stents and that intimal hyperplasia is the dominant contributor to late tissue prolapse.15 Because late lumen loss and loss index were higher in the patients treated with the GR-II stent, it is clear that there was either late mechanical recoil of the GR-II coil stent, increased intimal hyperplasia (or late tissue prolapse), or a combination of both.

**Study Limitations**

Limitations of the present study include the inherent differences in stent lengths between the GR-II and the PS stents, making a precise comparison problematic. In addition, inconsistencies in operator technique and infrequent optimal GR-II size selection resulted in pronounced intersite TLR variability. Because greater variability was noted in the GR-II–treated patients, a “roll in” learning phase may have reduced some of the observed differences. Perhaps these issues should serve as an important lesson for future randomized clinical trials. Appropriate interdevice comparisons require that operator technique factors relating to new devices be completely understood and consistently applied by investigators before embarking on definitive randomized controlled clinical trials. In this context, it may be appropriate for the FDA to require sponsors to demonstrate optimal device use and technique by the investigators before approving the initiation of large-scale randomized clinical trials and the release of new devices.

We conclude that in patients undergoing elective stent placement in de novo native coronary lesions, the GR-II coronary stent is associated with higher stent thrombosis, angiographic restenosis, and late TLR rates.

**Appendix**

The following investigators were participants in the clinical trial: Arizona Heart Institute and Foundation, Phoenix, R.R. Heuser; Montgomery Cardiovascular, Montgomery, Ala, P.B. Moore; Beth Israel Hospital, Boston, Mass, J.P. Carrozzi, Jr; Brigham and Women’s Hospital, Boston, Mass, J.A. Bittl; Lindner Center for Cardiovascular Research, Cincinnati, Ohio, D.J. Kereakes; The Cleveland Clinical Foundation, Cleveland, Ohio, S.G. Ellis; Duke North Hospital, Durham, NC, J.P. Zidar; North Ohio Heart Center, Elyria, C.D. O’Shaughnessy; Iowa Heart Center, Des Moines, L.A. Iannone; Johns Hopkins Hospital, Baltimore, Md, J.A. Brinker; Lenox Hill Hospital, New York, NY, J.W. Moses; Loma Linda Hospital, Los Angeles, Calif, C.E. Ruiz; Mayo Clinic Foundation, Rochester, Minn, D.R. Holmes; The Methodist Hospital, Houston, Tex, A.E. Raizner; Mid-Cardiology Cardiology, Charlotte, NC, D.A. Cox; Midwest Cardiology Research Foundation, Columbus, Ohio, S.J. Yakubov and B.S. George; Prairie Cardiovascular Consultants, Ltd, Springfield, Ill, K. Roche-Singh; Rhode Island Hospital, Providence, D.O. Williams; Rochester General Hospital, Rochester, NY, M.A. Thompson; Sacred Heart Hospital, Pensacola, Fla, E.W. Rogers; St. Joseph Hospital, Atlanta, Ga, W.D. Knoff; St. Luke’s Hospital, Milwaukee, Wis, G. Dorros; St. Vincent Hospital, Indianapolis, Ind, E.T.A. Fry and J.B. Hermiller; Sanger Clinic, PA, Charlotte, NC, C.A. Simonett; Summitt Cardiology, Seattle, Wash, D. Warth; Medical College of Ohio at Toledo, C.J. Cooper; University of Alabama at Birmingham, L.S. Dean and G.S. Roubin; University Hospital, Tampa, Fla, R.E. Bowerman; Vancouver Hospital and Health Science Center, Vancouver, BC, I.M. Penn; Washington Hospital Center, Washington, DC, M.B. Leon; and William Beaumont Hospital, Royal Oak, Mich, R.D. Safian.

**References**


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