

SCIENTIFIC LETTER

Impact of glycaemic and lipid control on outcome after percutaneous coronary interventions in diabetic patients

C Briguori, G Condorelli, F Airoidi, G W Mikhail, B Ricciardelli, A Colombo

Heart 2004;90:1481-1482. doi: 10.1136/hrt.2003.030437

Patients with type 2 diabetes mellitus account for approximately 20% of patients undergoing percutaneous coronary interventions (PCI). PCI is less effective in diabetic than in non-diabetic patients.^{1,2} Glycaemic and lipid control may have an impact on the clinical outcome in type 2 diabetic patients following elective PCI.

METHODS

From January 2000 to June 2001, 280 consecutive patients with type 2 diabetes mellitus successfully underwent their first elective PCI at one of our institutions. Optimal metabolic control was defined as a glycosylated haemoglobin (HbA1c) concentration of < 7.0% and a low density lipoprotein cholesterol (LDL-C) concentration < 100 mg/d (< 2.6 mmol/l). Three groups were identified: (1) optimal group (n = 45, 16% of patients) with optimal glycaemic and lipid control; (2) suboptimal group (n = 126, 45% of patients) with only one target value reached; (3) poorly controlled group (n = 109, 39% of patients) who failed to reach either glycaemic or lipid target values. The end point of the study was the rate of major adverse cardiovascular events (MACE), defined as death of any cause, non-fatal myocardial infarction, and target vessel revascularisation, at 12 months in the three groups of patients. All patients received aspirin (325 mg daily, indefinitely) and ticlopidine (250 mg twice daily, for at least 30 days) or clopidogrel (75 mg daily, for at least 30 days). Glycoprotein IIb/IIIa inhibitors were administered according to operator discretion.

RESULTS

Clinical, angiographic, and procedural characteristics are summarised in table 1. At mean (SD) 12 (4) months, MACE occurred in 6 (13.3%) patients in the optimal group, in 40 (32%) patients in the sub optimal group, and in 55 (50.5%) patients in the poorly controlled group (p < 0.001). Variables entered into the Cox regression analysis were: age \geq 70 years, sex, insulin treatment, statin treatment, nephropathy, optimal glycaemic and lipid control, small vessel, elective glycoprotein IIb/IIIa inhibitors, left ventricular ejection fraction < 40%, complete revascularisation, and multivessel PCI. The independent predictors of MACE at follow up were: insulin treatment (hazard ratio (HR) 3.43, 95% confidence interval (CI) 1.59 to 7.42; p = 0.002), optimal group (HR 0.29, 95% CI 0.09 to 0.97; p = 0.045), and age \geq 70 years (HR 2.10, 95% CI 1.10 to 4.25; p = 0.045).

DISCUSSION

We showed that an HbA1c concentration of < 7% and an LDL-C concentration of < 100 mg/dl favourably influenced the outcome in diabetic patients after PCI. A very low event rate was observed in patients with both a strict glycaemic and blood lipid control. Our study highlights shortcomings in the effectiveness of treatment and prevention of risk factors that are associated with high morbidity and mortality. It may be that a stricter surveillance, a better lifestyle, and a more aggressive pharmacological approach would allow us to improve metabolic control.^{3,4} The present study was not a randomised controlled

Table 1 Patient characteristics according to glycaemic and lipid control

| | Optimal group (n = 45) | Suboptimal group (n = 126) | Poorly controlled group (n = 109) | p Value |
|---|------------------------|----------------------------|-----------------------------------|---------|
| Age (years) | 63 (7) | 62 (9) | 62 (10) | 0.67 |
| Male | 39 (87%) | 104 (82.5%) | 74 (68%) | 0.008 |
| Unstable angina | 7 (16%) | 23 (18%) | 26 (24%) | 0.63 |
| Diabetes treatment | | | | 0.33 |
| Non-insulin requiring* | 36 (80%) | 102 (81%) | 80 (73%) | |
| Insulin requiring† | 9 (20%) | 24 (19%) | 29 (27%) | 0.14 |
| Statin treatment | 35 (78%) | 83 (66%) | 66 (61%) | 0.14 |
| HbA1c (%) | 6.1 (0.6) | 7.2 (1.4) | 8.5 (1.3) | <0.001 |
| LDL-C (mmol/l)‡ | 2.05 (0.42) | 2.63 (0.80) | 3.56 (0.68) | <0.001 |
| Left ventricular ejection fraction (%) | 58 (12) | 57 (11) | 57 (9) | 0.75 |
| Previous myocardial infarction | 25 (56%) | 65 (52%) | 50 (46%) | 0.58 |
| Previous bypass surgery | 4 (9%) | 20 (16%) | 10 (9%) | 0.34 |
| Systemic hypertension | 33 (73%) | 84 (67%) | 81 (74%) | 0.31 |
| Smoker | 21 (47%) | 69 (55%) | 40 (37%) | 0.025 |
| Nephropathy | 18 (40%) | 54 (43%) | 54 (50%) | 0.51 |
| Distribution of coronary artery disease | | | | 0.090 |
| Single vessel | 19 (42%) | 35 (28%) | 34 (31%) | |
| Double vessel | 19 (42%) | 51 (40.5%) | 36 (33%) | |
| Triple vessel | 7 (16%) | 40 (31.5%) | 39 (36%) | |
| Number of treated vessels/patient | 1.3 (0.5) | 1.3 (0.5) | 1.3 (0.5) | 0.96 |
| Number of treated lesions/patient | 1.6 (0.8) | 1.5 (0.7) | 1.6 (0.9) | 0.49 |
| Diameter stenosis (%) | | | | |
| Pre- | 83 (13) | 82 (16) | 79 (15) | 0.22 |
| Post- | 6 (10) | 5 (9) | 8 (14) | 0.12 |
| Reference vessel diameter (mm) | | | | |
| Pre- | 2.91 (0.58) | 2.88 (0.60) | 2.95 (0.59) | 0.37 |
| Post- | 3.04 (0.61) | 3.05 (0.61) | 3.08 (0.64) | 0.71 |
| Lesion length (mm) | 11 (5) | 11 (5) | 11 (4) | 0.75 |
| Complete revascularisation | 22 (49%) | 59 (47%) | 55 (50.5%) | 0.57 |
| Type of PCI | | | | 0.93 |
| Balloon only | 4 (9%) | 11 (9%) | 11 (10%) | |
| Bare metal stent | 41 (91%) | 115 (91%) | 98 (90%) | |
| Elective glycoprotein IIb/IIIa inhibitors | 17 (38%) | 46 (37%) | 44 (41%) | 0.79 |

*Non-insulin requiring which includes patients treated with diet and oral hypoglycaemic drugs but no insulin.

†Insulin requiring which includes patients treated with insulin, regardless of other treatment.

‡To convert values of LDL-C to mg/dl, divide by 0.026.

trial. We cannot therefore exclude the fact that patients with poorer glycaemic and lipid profiles were in a more advanced stage of coronary atherosclerosis. Furthermore, the potential impact of glycoprotein IIb/IIIa antagonists and statins may not be apparent as the study was not powered to address this.

Abbreviations: HbA1c, glycosylated haemoglobin; LDL-C, low density lipoprotein cholesterol; MACE, major adverse cardiovascular events; PCI, percutaneous coronary intervention

Authors' affiliations

C Briguori*, **B Ricciardelli**, Laboratory of Interventional Cardiology, Clinica Mediterranea, Naples, Italy

F Airoidi, **G W Mikhail**, **A Colombo**, San Raffaele Hospital, Milan, Italy

G Condorelli, Department of Biology, Cellular and Molecular Pathology, "Federico II" University, Naples, Italy

*Also at San Raffaele Hospital, Milan, Italy

Correspondence to: Dr Carlo Briguori, Interventional Cardiology, Clinica Mediterranea, Via Orazio 2, I-80121, Naples, Italy; briguori.carlo@hsr.it

Accepted 16 February 2004

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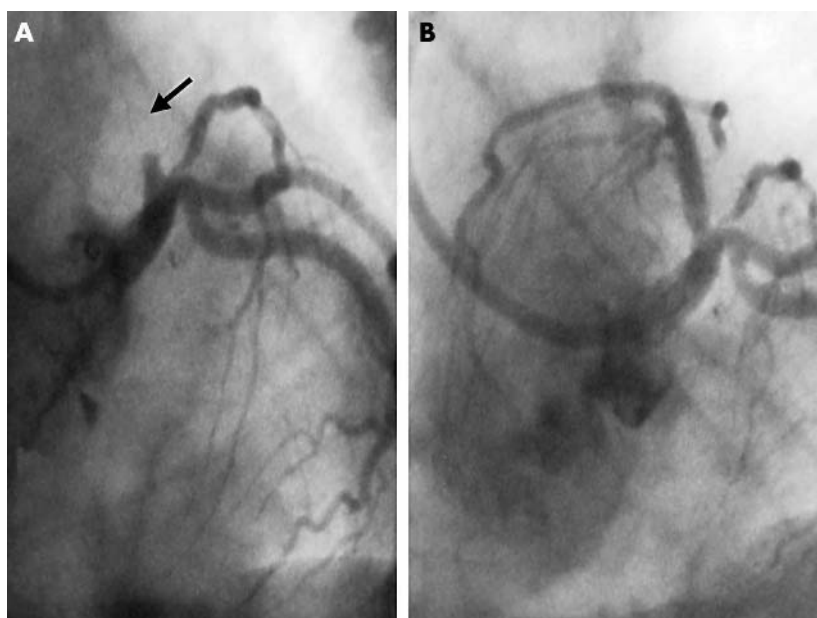
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doi: 10.1136/hrt.2004.033589

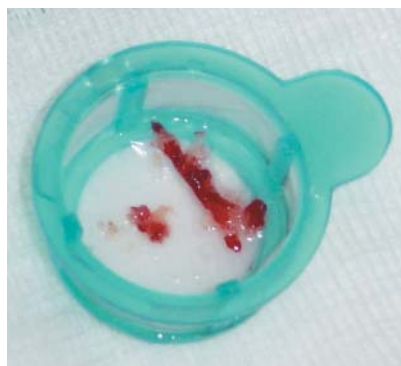
Late thrombotic occlusion of paclitaxel eluting stent more than one year after stent implantation

Use of drug eluting stents has become the state-of-the-art therapy for percutaneous treatment of obstructive coronary artery disease. However, concern has been expressed on the delayed healing after implantation of a drug eluting stent. We present the first report of late thrombotic occlusion of a paclitaxel eluting stent (Taxus, Boston Scientific Corp) at just more than one year after implantation.

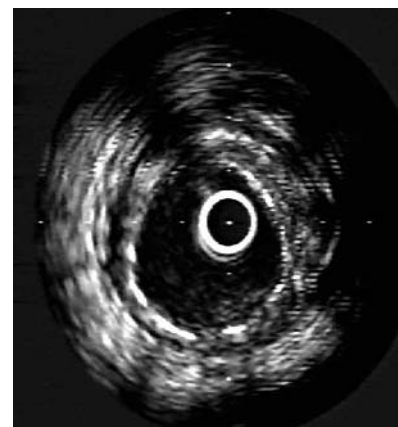
A 44 year old Indian man underwent implantation of a 3.0/24 mm paclitaxel eluting stent on 27 September 2002 for treatment of unstable angina caused by proximal left anterior descending (LAD) artery stenosis. Long term aspirin and six months of clopidogrel were prescribed, together with anti-lipid treatment. The patient was compliant but continued to smoke. He remained well until 8 October 2003 when he presented with chest pain for three hours, associated with diaphoresis. A 12 lead ECG showed ST segment elevation consistent with acute anterior myocardial infarction. Emergency coronary angiography confirmed a totally occluded proximal LAD artery, the site of the paclitaxel eluting stent (panel A). The occlusion was crossed using a 0.014 inch floppy guidewire, with immediate restoration of antegrade TIMI grade 2 flow. Heavy thrombus burden was noted in-stent. A PercuSurge Export aspiration catheter was used to aspirate the thrombus (lower left panel). Intravascular ultrasound (lower right panel) showed only mild in-stent neointimal hyperplasia, suggesting primarily a thrombotic event. The stent was well expanded except for a small segment at the proximal stent edge. No stent malapposition or edge stenosis was noted. A 3.0/10 mm cutting balloon, and then a 3.25/15 mm NC monorail balloon was used to dilate the stent in sequence. The procedure was performed with the support of double bolus intracoronary doses of eptifibatide, given 10 minutes apart, followed by continuous intravenous infusion for 48 hours. Final angiography (panel B) showed < 10% residual stenosis, TIMI 3 flow and myocardial blush grade 3



(A) Diagnostic angiography showed total occlusion of the paclitaxel eluting stent (arrow). (B) Final angiography after thrombus aspiration and balloon dilatation of the stent showed normal antegrade flow (TIMI 3).



Thrombus aspirated by the PercuSurge Export aspiration catheter.



Intravascular ultrasound image of the stent after thrombus aspiration. The stent was well expanded and apposed. No significant in-stent neointimal hyperplasia was detected.

C H Lee
H C Tan
H Y Ong
S G Teo
Y T Lim

mdclimyt@nus.edu.sg