

Second-generation Drug-eluting Stenting vs Coronary Artery Bypass Grafting for Coronary CTO

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Background/Aims: Limited data are available regarding the long-term clinical outcomes of percutaneous coronary intervention (PCI) using second-generation drug-eluting stent (DES) versus coronary artery bypass grafting (CABG) in patients with coronary chronic total occlusion (CTO). We compare the long-term clinical outcomes of patients with CTO and multivessel coronary artery disease (CAD) treated with PCI using second-generation DES versus CABG. **Methods:** Between January 2008 and February 2012, we analyzed data from 423 consecutive patients undergoing successful revascularization for CTO and multivessel CAD. Patients treated with PCI using second-generation DES (n=232, 2nd DES-PCI group) were compared with those treated with CABG (n=191, CABG group) in terms of death or myocardial infarction (MI) and major adverse cardiac and cerebrovascular event (MACCE). Inverse probability of treatment weighting (IPTW) was also performed. **Results:** During a median follow-up duration of 30 months, in multivariate analysis, there was no significant difference of death or MI (hazard ratio [HR]: 0.97; 95% confidence interval [CI]: 0.39 to 2.39; $p=0.939$) and MACCE (HR: 1.03; 95% CI: 0.53 to 2.01; $p=0.938$) between the 2nd DES-PCI group and the CABG group. After IPTW adjustment, the incidence of death or MI (HR: 0.97; 95% CI: 0.39 to 2.39; $p=0.939$) and MACCE (HR: 1.03; 95% CI: 0.53 to 2.01; $p=0.938$) were still similar in both two groups. **Conclusions:** The efficacy of PCI using second-generation DES was comparable to that of CABG in patients with CTO and multivessel CAD.

Table 1. Baseline Clinical Characteristics

	2nd DES group n = 232	CABG group n = 191	p value		2nd DES group n = 232	CABG group n = 191	p value
Age (year)	63.1 ± 11.1	62.9 ± 9.9	0.821	CTO location			
Male	186 (80.2)	160 (83.8)	0.340	RCA	96 (41.4)	118 (61.8)	<0.001
Diabetes mellitus	97 (41.8)	112 (58.6)	0.001	LM	0 (0)	9 (0)	
Hypertension	111 (47.8)	122 (63.9)	0.795	LAD	92 (39.7)	68 (35.6)	0.392
Dyslipidemia	118 (50.9)	70 (36.6)	0.003	Lcx	77 (33.2)	83 (43.5)	0.030
Current smoker	77 (33.2)	57 (29.8)	0.462	Multiple CTOs	50 (21.6)	70 (36.6)	0.001
Chronic renal failure	16 (6.9)	17 (8.9)	0.444	Abrupt stump	67 (28.9)	88 (46.1)	<0.001
Acute coronary syndrome	82 (35.3)	34 (17.8)	<0.001	Bridge collaterals	56 (24.1)	85 (44.5)	<0.001
Previous MI	33 (14.2)	31 (16.2)	0.567	Calcified CTO	28 (12.1)	55 (28.8)	<0.001
Previous CVA	15 (6.5)	15 (7.9)	0.580	*Proximal to mid CTO	169 (72.8)	139 (72.8)	0.987
Previous PCI	43 (18.5)	30 (15.7)	0.444	**Well-developed collateral	75 (46.0)	82 (42.9)	0.561
LVEF (%)	56.5 ± 13.1	52.0 ± 14.3	0.001	SYNTAX score	22.2 ± 9.5	29.8 ± 10.6	<0.001
Concomitant medications				SYNTAX score ≥24	66 (40.5)	131 (68.6)	<0.001
Aspirin	222 (95.7)	172 (90.1)	0.022	Data are presented as n (%). CI = confidence interval.			
Statins	185 (79.7)	149 (78.0)	0.664	*Well-developed "CTO of the proximal to middle portions of the vessel" as "Proximal to mid CTO"			
Beta blockers	141 (60.8)	14 (7.4)	0.001	**Well-developed "Rancho grade 3 collateral flow" as "Well-developed collateral"			
ACE inhibitors/ARBs	140 (60.3)	35 (18.3)	<0.001	CABG = coronary artery bypass grafting; CVA = cerebrovascular accident; DES = drug-eluting stent; LVEF = left ventricular ejection fraction; MI = myocardial infarction; PCI = percutaneous coronary intervention.			

Table 2. Angiographic Characteristics

	2nd DES group n = 232	CABG group n = 191	p value
Death or MI	8 (3.4)	13 (6.8)	0.399
Death	6 (2.6)	13 (6.8)	0.165
Cardiac death	2 (0.9)	6 (3.1)	0.259
MI	3 (1.3)	1 (0.5)	0.202
CVA	3 (1.3)	10 (5.2)	0.077
Repeat revascularization	20 (8.6)	1 (0.5)	0.004
MACCE	28 (12.1)	21 (11.0)	0.341

Table 3. Clinical outcomes for adjusted hazard ratios and Inverse Probability-of-Treatment Weighting Method

	2nd DES group n = 232	CABG group n = 191	adjusted HR (95% CI)	p value	HR after IPTW (95% CI)	p value
Death or MI	8 (3.4)	13 (6.8)	0.69 (0.29 - 1.63)	0.399	0.72 (0.26 - 1.95)	0.518
Death	6 (2.6)	13 (6.8)	0.32 (0.20 - 1.31)	0.165	0.54 (0.18 - 1.63)	0.278
Cardiac death	2 (0.9)	6 (3.1)	0.45 (0.11 - 1.81)	0.259	0.39 (0.07 - 2.15)	0.281
MI	3 (1.3)	1 (0.5)	4.28 (0.46 - 39.74)	0.202	2.94 (0.22 - 39.83)	0.418
CVA	3 (1.3)	10 (5.2)	0.31 (0.08 - 1.15)	0.077	0.60 (0.15 - 2.44)	0.477
Repeat revascularization	20 (8.6)	1 (0.5)	19.56 (2.59 - 147.82)	0.004	28.66 (3.50 - 233.06)	0.002
MACCE	28 (12.1)	21 (11.0)	1.32 (0.74 - 2.35)	0.341	1.49 (0.76 - 2.91)	0.244

Data are presented as n (%).
 Adjusted covariates include diabetes mellitus and SYNTAX score ≥24.
 MACCE was defined as a composite of death, MI, CVA or repeat revascularization.
 CABG = coronary artery bypass grafting; CI = confidence interval; CVA = cerebrovascular accident; DES = drug-eluting stent; HR = hazard ratio; IPTW = inverse probability of treatment weighting; MI = myocardial infarction; MACCE = major adverse cardiac and cerebrovascular event; SYNTAX = the Syntax System for PCI with Taux and Cardiac Surgery.

Early diagnosis of Iron overloads cardiomyopathy with magnetic resonance imaging T2*

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A 43-year-old female refer to the general surgery with dyspnea and elevated cardiac enzymes. She received a liver transplant two months ago and was taking immunosuppression agents. Transthoracic echocardiography showed a mildly dilated left ventricle (LV) and global hypokinesia with an LV ejection fraction of 29.3%. LV contractile function has been decreased compared to baseline study. (EF 61.7 -> 29.3%). Coronary angiography was normal and we performed treatment for heart failure. Patient tacrolimus trough concentration was 23.4 ng/mL (range 5-20 ng/mL), the drug suspected to cause cardiomyopathy, was discontinued and replaced with Everolimus. Magnetic resonance imaging was performed to confirm the tacrolimus induced cardiomyopathy. But LV myocardium appeared diffusely dark on gradient-echo sequences with T2* (T2 star), consistent with severe myocardial iron deposition. (Figure 1). Dark signal intensity of the liver region of interest(ROI), gradient-echo sequences with T2* consistent with severe iron overload of these organs. Laboratory studies revealed: serum ferritin 4,982 ng/mL (13-150ng/mL) and iron levels of 230 mcg/dL (50-150 mcg/dL). We considered chelation therapy but improved with deferasirox therapy. Our case study shows that cardiac MRI sequences may assist in the initial diagnosis of cardiomyopathy due to iron deposition. We conclude that cardiac MRI can be used as a non-invasive alternative to biopsy in patients with iron overloads cardiomyopathy.

