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TOPIC HIGHLIGHT

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Chronic total occlusion: To treat or not to treat

Alfredo Bardají, Judit Rodriguez-López, Mauricio Torres-Sánchez

Alfredo Bardají, Judit Rodriguez-López, Mauricio Torres-Sánchez, Servicio de Cardiología, Hospital Universitario de Tarragona Joan XXIII, Universitat Rovira Virgili, 3007 Tarragona, España

Author contributions: Bardají A, Rodriguez-López J and Torres-Sánchez M performed research; Bardají A wrote the paper.

Correspondence to: Alfredo Bardají, MD, PhD, FESC, Servicio de Cardiología, Hospital Universitario de Tarragona Joan XXIII, Universitat Rovira Virgili, Calle Dr Mallafré Guasch 4, 43007 Tarragona, España. alfredo.bardaji@urv.cat

Telephone: +34-97-7295834 Fax: +34-97-7295859

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Abstract

Over the last two decades, there has been increasing interest in new techniques for the percutaneous treatment of coronary chronic total occlusions (CTO), which have a success rate that is much higher than that of a few years ago. The rise in percutaneous treatment for these lesions is due to its ability to improve the symptoms and prognosis of patients in the chronic and stable phase of coronary disease. Current data suggest that successful percutaneous coronary intervention for CTO is associated with improvement in patient symptoms, quality of life, left ventricular function, and survival, compared with those with unsuccessful CTO PCI. However, all the scientific evidence supporting this treatment comes from observational studies, and no randomized study comparing percutaneous treatment with medical treatment has yet been published. A major limitation of these studies is their observational design, with limited information with regard to potential baseline differences between the successful *vs* unsuccessful cohorts. Pending randomized studies, patients should be selected very carefully, especially if they are asymptomatic or very few symptoms, and the benefits obtained in terms of complications during the procedure, the quality of life obtained and further ischemic events avoided should be evaluated systematically. In this review, we will consider the available information supporting percutaneous treatment for chronic occlusions, as well as the areas of uncertainty where more research projects are required.

Key words: Chronic total occlusion; Percutaneous coronary intervention

Core tip: This is a critical review about the available information supporting percutaneous treatment for chronic occlusions, as well as the areas of uncertainty where more research projects are required.

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INTRODUCTION

Chronic total occlusions (CTO) are considered to be 100% coronary lesions, of more than 3 mo evolution[1]. They are therefore always found in stable chronic patients, with varying levels of symptoms. After the culprit artery has been treated, patients with acute coronary syndrome may occasionally also have a chronic occlusion in another artery that was not responsible for the acute event, and is therefore considered a CTO.

DEFINITION AND INCIDENCE

The prevalence of CTO in patients undergoing coronary angiography varies, ranging between 18% and 52% depending on the clinical profile of the patient being examined[2-7] (Table 1). Although revascularization surgery is the most frequent treatment, clinicians and invasive cardi-

Table 1 Chronic total occlusion prevalence, location and treatment applied in different studies *n* (%)

Ref.	Type of study	Population	CTO prevalence	CTO location			Medical treatment	PCI	CABG
				RCA	LAD	LCA			
Kahn *et al*[2], 1993	Retrospective	333 Coronary disease (stenoses ⩾ 50%)	101 (35)	58%	18%	24%	-	-	-
Christofferson *et al*[3], 2005	Retrospective	6581 Underwent coronarography because of suspected CD	1612 (25)	49.4%	22%	28.60%	49%	11%	40%
		3087 Coronary disease (stenoses ⩾ 70%)	1612 (52)						
Srinivas *et al*[4], 2002	Retrospective	1761 Multivessel disease	545 (31)	-	-	-	-	14.50%	-
Yamamoto *et al*[5], 2013	Prospective	15263 First revascularization procedure	2491 (19)	44.9%	41.10%	28.50%	-	61.18%	-
Fefer *et al*[6], 2012	Prospective	14439 Underwent coronariography because of suspected CD	2630 (18.2)	46.9%	19.86%	15.43%	64%	10%	26%
Jeroudi *et al*[7], 2013	Prospective	1015 Coronary disease (stenoses ⩾ 50%)	319 (31.34)	-	-	-	19% (61)	50% (161)	30% (97)

CTO: Chronic total occlusion; RCA: Right coronary artery; LAD: Left anterior descending; LCA: Left circumflex artery; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass graft.

ologists often consider the need and feasibility of percutaneous treatment for these lesions, based on symptoms and prognostic factors. However, as it is a common problem in all Cath Labs, the extensive variability between different centres is striking. For example, in North American centres[4], with an incidence rate of CTO of between 29% and 33% in all the catheterizations performed, only between 6% and 9% of patients were treated percutaneously. However, in Japanese centres, with an incidence of 19% of CTO in all the catheterizations performed, 61.2% of all cases were treated percutaneously[5]. There are also significant differences in the treatment of CTO within the same geographical area or healthcare system. For example, in the Canadian CTO registry[6], some hospitals percutaneously treat 16% of their patients, while others only do so for 1%. These differences are very striking, and can only be justified by some generally ill-defined indications, as well as the technical difficulty that means that not all invasive cardiologists can or should deal with complex lesions of this type. However, there is another factor that also needs to be mentioned. Patients with CTO probably have a clinical profile that is different to that of patients with chronic coronary ischemic disease in general. There not only are differences in terms of greater severity of coronary disease, but also in terms of increased non-coronary comorbidity, such as a higher rates of prevalence of diabetes, peripheral arterial disease, heart failure and a history of strokes[8]. The indications for percutaneous treatment of CTO are not well defined in the European guidelines for revascularization[9], or in the guidelines for patients with stable chronic coronary disease[1] (Table 2). The American guidelines on revascularization[10,11] and chronic stable ischemic heart disease[12] are also unclear as regards the indications for treatment of CTO. Only the American guidelines for the appropriate use of percutaneous coronary treatment[13] contain a clear position on treatment that is appropriate, uncertain or not indicated in CTO lesions (Table 3).

CTO TREATMENT IN PATIENTS WITH ANGINA

There should be no doubt that a treatment of a CTO affecting an ischemic myocardial area that causes symptoms such as angina should improve patients' symptoms, by providing a greater perfusion flow than that provided by collateral circulation, as a consequence of opening the occluded artery[14]. However, this has been poorly studied and quantified in the medical literature. Very few studies have specifically evaluated the changes in the ischemic threshold and quality of life scales of symptomatic and asymptomatic patients with percutaneously treated CTO. In the FACTOR Trial (FlowCardia Approach to CTO Recanalization), 125 patients completed the Seattle Angina Questionnaire at baseline and one month after percutaneous coronary intervention[15]. Successful treatment was associated in overall terms with an improvement in the frequency of angina, physical capacity and quality of life. However, this improvement was only observed in previously symptomatic patients but not in asymptomatic patients. In fact, this symptomatic improvement is similar to that obtained with percutaneous coronary intervention in the treatment of lesions without chronic total occlusion[16].

TREATMENT OF CTO IN ISCHEMIC PATIENTS

Often no distinction is made between patients with angina and patients with myocardial ischemia when percutaneous treatment of CTO is indicated[17]. However, these two concepts are different in our opinion, and should be clarified. In patients with angina (and therefore with

Table 2 Specific recommendations on the treatment of chronic total occlusion in the American and European Practice Guidelines

Society	Guideline	Specific recommendation on the treatment of CTO
EUROPEAN	2010 Guidelines of myocardial revascularization[9]	"Revascularization of CTO may be considered in the presence of angina or ischemia related to the corresponding territory"
	2013 ESC guidelines on the management of stable coronary artery disease[1]	"Revascularization needs to be discussed in patients with symptoms of occlusion or large ischemic areas"
AMERICAN	2011 ACCF/AHA Guideline for Coronary Artery Bypass Graft Surgery[10]	Not mentioned
	2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention[11]	Recommendation IIa. Evidence level B. PCI of a CTO in patients with appropriate clinical indications and suitable anatomy is reasonable when performed by operators with appropriate expertise "The decision to try PCI for a CTO (*vs* continued medical therapy or surgical revascularization) requires an individualized risk-benefit analysis encompassing clinical, angiographic, and technical considerations"
	2012 ACCF/AHA/ACP/AATS/PCNA/SCAI/STS Guideline for the Diagnosis and Management of Patients With Stable Ischemic Heart Disease[12]	Not mentioned

CTO: Chronic total occlusion; ACCF: American College of Cardiology Foundation; AHA: American Heart Association; SCAI: Society for Cardiovascular Angiography and Interventions; ACP: American College of Physicians; AATS: American Association for Thoracic Surgery; PCNA: Preventive Cardiovascular Nurses Association; STS: Society of Thoracic Surgeons; PCI: Percutaneous Coronary Intervention.

Table 3 Specific recommendations on the treatment of chronic total occlusion in the 2012 ACCF/SCAI/STS/AATS/ASNC/HFSA/SCCT Appropriate Use Criteria for Coronary Revascularization Focused Update[14]

		ANGINA						
		Asymptomatic	I	II	III	IV		
Risk in the ischemia test	High	Uncertain	Appropriate	Appropriate	Appropriate	Appropriate	Max	Treatment level
		Uncertain	Uncertain	Uncertain	Appropriate	Appropriate	Med	
		Uncertain	Uncertain	Uncertain	Appropriate	Appropriate	Min	
	Medium	Uncertain	Uncertain	Uncertain	Appropriate	Appropriate	Max	
		Inappropriate	Uncertain	Uncertain	Uncertain	Uncertain	Med	
		Inappropriate	Uncertain	Uncertain	Uncertain	Uncertain	Min	
	Low	Inappropriate	Inappropriate	Inappropriate	Uncertain	Uncertain	Max	
		Inappropriate	Inappropriate	Inappropriate	Inappropriate	Inappropriate	Med	
		Inappropriate	Inappropriate	Inappropriate	Inappropriate	Inappropriate	Min	

It shows the 45 possible scenarios depending on the risk of mortality based on the findings on ischemia tests, symptoms and level of treatment.

ischemia), the benefit of CTO treatment is for the symptoms and possibly the prognosis. However, as mentioned above, in patients with ischemia but without angina, the benefit is not symptomatic and can only be evaluated in prognostic terms. It is therefore important to determine whether patients with myocardial ischemia but who are asymptomatic benefit from percutaneous treatment of a CTO. The rationale for this approach is based on relatively early studies in which the improvement of ischemia provided by the revascularization obtained by an angioplasty, in both symptomatic patients[18] and asymptomatic patients[19], was associated with an improved prognosis. In the SWISSI II Trial[19] in the late 1990s, on asymptomatic patients after myocardial infarction, with coronary disease in 1 or 2 vessels and inducible myocardial ischemia in an imaging stress test, coronary angioplasty significantly reduced coronary events during a long-term follow-up period. However, in this study, both the medical treatment, which was very limited, and the percutaneous treatment (the use of bare metal stents) were obviously different to those currently in use. More recent studies of symptomatic patients with chronic coronary disease, frequently presenting a positive test for ischemia, have failed to show that percutaneous revascularization improves prognosis[20], even in diabetic patients[21]. In the COURAGE trial, the small benefit in terms of improved quality of life in percutaneously treated patients compared to those receiving medical therapy without revascularization disappeared after 36 mo follow-up[22]. The data from the COURAGE trial substudy, with quantification of ischemia by a stress test with nuclear imaging, show that in patients with stable chronic ischemic heart disease, angioplasty provides a greater improvement in the ischemic area than medical treatment[23]. However, this improvement in the ischemic area had no effect on the medium-term prognosis[24]. A recent meta-analysis including all the randomized studies in patients with stable chronic ischaemic cardiopathy and proven myocardial ischemia concluded that percutaneous treatment does not affect rates of mortality, myocardial infarction, unplanned revascularization or angina compared to medical treatment alone[25]. At present, the hypothesis that moderate to severe

Table 4 Findings on left ventricular ejection fraction and regional wall motion variations after percutaneous coronary intervention treatment of chronic total occlusion

	Type of study	Population	LVEF estimation	Follow up	Results				
					LVEF	Regional wall motion	Symptoms	Collateral function	Ventricular remodeling
1994-1995 Sirnes *et al*[30]	Prospective	95 CTOs treated with PCI	Ventriculography	Angiography 6 mo	LVEF increase (from 0.62 ± 0.13 to 0.67 ± 0.12) $P < 0.001$	Increase in regional radial shortening (from 0.279 ± 0.106 to 0.319 ± 0.107) $P < 0.001$	Improvement in angina class	Not mentioned	Not mentioned
1999-2003 Werner *et al*[31]	Prospective	126 CTOs treated with PCI	Ventriculography	Angiography	LVEF increase (from 0.60 ± 0.19 to 0.67 ± 0.16) $P < 0.001$	Increase in wall motion severity index (from -1.92 ± 1.32 to -1.30 ± 1.28) $P < 0.001$	Not mentioned	No changes in collateral function	Not mentioned
2008 Kirschbaum *et al*[32]	Prospective	21 CTOs treated with PCI	NMR	NMR 5 mo and 3 yr	LVEF increase (from 60% ± 9% to 63% ± 11%) $P = 0.11$	Increase in segmental wall thickening. From 19% ± 21% to 31% ± 30% at 5 mo ($P < 0.001$) and 47% ± 46% at 3 yr ($P = 0.04$)	Not mentioned	Not mentioned	Less ventricular remodeling in NMR at 3 yr

LVEF: Left ventricular ejection fraction; CTO: Chronic total occlusion; PCI: Percutaneous coronary intervention; NMR: Nuclear magnetic resonance.

myocardial ischemia should be revascularized in order to improve the prognosis must therefore be reviewed[26]. In the context of patients with chronic coronary artery disease, the ISCHEMIA clinical trial will attempt to demonstrate whether the strategy of cardiac catheterization for revascularization is better than strategy of medical treatment in patients with moderate to severe ischemia detected in a stress test with imaging[27]. In this trial, there will presumably be few patients with CTO, meaning that it is possible that its findings cannot be fully extrapolated to this specific population. As regards patients specifically with CTO, there are two ongoing clinical trials that are randomizing patients with angina or ischemia in an imaging test for medical treatment or angioplasty. The EURO-CTO clinical trial, being run at a European level, has the primary objective of evaluating quality of life at 12 mo, as well as assessing major coronary events after 3 years[28]. The DECISION-CTO clinical trial, conducted in Asian countries, has a composite primary endpoint (cardiac death, myocardial infarction, stroke or further revascularization) evaluated after 3 years[29].

CTO TREATMENT IN PATIENTS WITH VENTRICULAR DYSFUNCTION

Chronic hypoperfusion due to the presence of a CTO on a viable myocardium can cause ventricular dysfunction, and may lead to symptoms such as exercise intolerance and heart failure resulting from this dysfunction. It therefore seems logical that the opening of an occluded artery which irrigates a viable but dysfunctional myocardium could reverse this dysfunction and improve these patients' symptoms and prognosis. There are few studies, all of which are observational, that have specifically addressed this issue (Table 4). Most available data suggest a very modest improvement in ventricular function as a result of opening an occluded artery. For example, Sirnes assessed the changes in ventricular function by ventriculography and was only able to demonstrate a 2% increase in ejection fraction, although the regional radial shortening increased by 16% in the revascularized areas[30]. This slight improvement in regional ventricular function does not appear to depend on the presence of pre-existing collaterals, but probably on preserved microvascular integrity[31]. The use of more accurate methods for quantifying ventricular function, such as cardiac magnetic resonance imaging, also confirms that the improvement in ventricular function as a result of opening an occluded artery is very modest[32]. The improvement in the prognosis of patients with ventricular dysfunction due to revascularization is currently a topic of heated debate, following the results of randomized STICH study[33]. In this clinical trial, patients with multivessel disease and ventricular dysfunction did not improve their prognosis as a result of revascularization surgery, in comparison with the medically treated group. Surprisingly, even the specifically studied patients with myocardial viability did not benefit from revascularization[34]. The STICH study did not include patients with CTO, but the concept and the comments are relevant, because the percutaneous treatment of CTO is often justified simply on the basis of viability.

Meanwhile, the treatment of a CTO as a cause of deterioration in the ejection fraction due to complications

Table 5 Baseline characteristics of clinical and angiographic variables in studies included on Joyal meta-analisis[42]

Ref.	Age (yr)		Male sex (%)		Multivessel disease (%)		Diabetes (%)		LVEF (%)		NYHA class 3-4 (%)		Renal dysfunction (%)		Occlusion length (mm)		Calcified vessel (%)		Ischemic burden	
	Success	Failure	Success	Failure	Success	Failure	Success	Failure	Success	Failure	Success	Failure	Success	Failure	Success	Failure	Success	Failure	Success	Failure
Finci *et al*[42], 1990	55 ± 11	55 ± 12	93	88	24	23	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d
Warren *et al*[43], 1990	54	55	53	47	48	52	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d
Ivanhoe *et al*[44], 1992	55 ± 10	56 ± 11	81	82	30	54 (0.0001)	10	15	55 ± 10	56 ± 11	3	3	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d
Angioï[45], 2000	55 ± 10	56 ± 11	52	88	37	45	10	11	59 ± 14	59 ± 14	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d
Noguchi *et al*[46], 2000	61 ± 9	61 ± 11	78	80	47	67 (0.01)	26	32	56 ± 12	54 ± 9	n/d	n/d	n/d	n/d	11.3 ± 8.3	14.1 ± 8.1 (< 0.05)	37	56 (< 0.01)	n/d	n/d
Suero *et al*[47], 2001	60 ± 11	61 ± 12	78	80	73	82 (0.001)	21	20	51 ± 14	52 ± 14	n/d	n/d	8.2	7.1	n/d	n/d	n/d	n/d	n/d	n/d
Olivari *et al*[48], 2003	58 ± 10	59 ± 11	86	85	45	60 (0.014)	17	20	56 ± 10	56 ± 10	9	7	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d
Hoye *et al*[49], 2005	60 ± 11	61 ± 10	74	72	54	67(0.03)	12	9.1	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d
Drozd *et al*[50], 2006	57 ± 10	58 ± 10	81	80	46	53	11	11	n/d	n/d	14.4	18 (NYHA ⩾ 2)	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d
Aziz *et al*[51], 2007	59	59	76	81	50	40 (0.006)	14	9	53	53	12.2	15.7	0.3	1.8	n/d	n/d	n/d	n/d	n/d	n/d
Prasad *et al*[52], 2007	63 ± 11	64 ± 11	76	75	70	74	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d	n/d
Valenti *et al*[53], 2008	67 ± 11	70 ± 11	81	83	85	87	24	21	42 ± 13	41 ± 14	n/d	n/d	n/d	n/d	25 (15-52.5)	28 (21-47.5)	n/d	n/d	n/d	n/d
de Labriolle *et al*[54], 2008	61 ± 12	64 ± 10	72	87	45	66 (0.002)	19	40.5 (0.005)	50 ± 12	48 ± 15	n/d	n/d	9.1	6.3	n/d	n/d	n/d	n/d	n/d	n/d

LVEF: Left ventricular ejection fraction; n/d: No data; NYHA: New York Heart Association.

during the procedure should not be ruled out. In recent years, major breakthroughs have been described in the material used for the percutaneous treatment of CTO, which has led to a significant reduction in complications[35]. However, the statement that today's complication rates are similar to those occurring in the treatment of less complex lesions could not be further from the truth. The Multinational CTO Registry mentions a rate of residual coronary dissection and perforation of 4.3% and 1.7% in successfully patients treated. However, among patients treated without success, these rates are 9.4% and 7.4%, respectively[35]. In the series from a large Japanese centre, the overall rate of perforation in all percutaneous coronary intervention procedures is 1.2%, but 44% of these occur in patients with CTO[36]. In another large Japanese centre, the rates of coronary dissection, perforation, distal embolization are 14.7%, 8.2% and 3.7% respectively, when a antegrade approach is used, and 10.1%, 13% and 1.4% respectively, when the procedure is performed *via* the retrograde route[37]. Some authors have postulated that this high rate of complications in unsuccessfully treated patients partially explains their worse prognosis compared with those who are successfully treated[38,39].

CTO TREATMENT TO IMPROVE PROGNOSIS

Some registries have reported that patients with complete revascularization have a better prognosis than those with incomplete revascularization, including the presence of

an untreated CTO[40]. On this basis, the main argument which normally supports the treatment of CTO is that successfully treated patients have a better prognosis than those treated without success. This is apparent in the Joyal meta-analysis, in which successful treatment was associated with a significant improvement in mortality compared to unsuccessfully treated patients[41]. This meta-analysis, conducted on studies with mainly retrospective data, seems to suggest that the baseline characteristics of successfully treated patients are similar to those treated without success, and that the unsuccessfully treated patients act as a medically treated control group. However, the studies performed with retrospective data[42-54] often lack information on some of the baseline characteristics of patients which have a clear effect on prognosis (Table 5). When studies with prospectively collected data are analyzed, it becomes apparent that the baseline characteristics of patients treated without success are clearly different from those who are successfully treated. For example, the Canadian prospective registry contains many variables of poor prognosis among the unsuccessfully treated patients, such as having a longer history of prior infarction, prior multivessel disease, a longer CTO, higher rate of residual dissection and perforation during the procedure, which undoubtedly influences the these patients' poor prognosis[36]. Furthermore, when the collection of variables is prospective and they are included in the predictive statistical model[55], the benefit of successful treatment of CTO is cancelled out as these patients have baseline characteristics with a better prognosis than those treated without success. This hypothesis is corroborated by the recent publication of the long-term results of patients in the CREDO-Kyoto Registry[5]. In this large series, the clinical evolution of 1192 successfully treated patients was compared with 332 unsuccessfully treated patients. Hospital mortality tended to be lower among the successfully treated patients than among those who were unsuccessfully treated (1.4% *vs* 3%, $P = 0.053$). During a three-year follow-up period, all-cause mortality did not differ between the two groups (9% *vs* 13.1%, $P = 0.18$), while the incidence of cardiac-related death was significantly lower in the successfully treated group (4.5% *vs* 8.4%, $P = 0.03$). However, after adjustment for confounding variables, successful treatment was not associated with either reduced total mortality (hazard ratio 0.93, 95%CI: 0.64 to 1.37, $P = 0.69$) or cardiac mortality (HR = 0.71, 95%CI: 0.44-1.16, $P = 0.16$). The only benefit associated with success in the treatment of CTO was a lower rate of surgical revascularization.

One group of patients deserves special consideration. These are patients with acute coronary syndrome in which the culprit artery is treated initially, but who have another chronically occluded artery which is considered for recanalization in a second procedure. This argument is based on the fact that these patients have a worse prognosis than patients with acute coronary syndrome with no CTO[56]. The EXPLORE clinical trial, which randomizes patients with CTO with no culprit artery after an acute coronary artery syndrome on revascularization treatment within 7 d of the ischemic event *vs* medical treatment[57] attempts to clarify this important issue, which is currently performed frequently without any scientific evidence.

CONCLUSION

Treatment of CTO has emerged in recent years as a result of a revolution in medical equipment that enables these patients to be managed with success rates well above those of a few years ago. However, there is an urgent need for randomized studies to support this therapy as it is not risk-free, and is very expensive. Pending randomized studies, patients should be selected very carefully, especially if they are asymptomatic or very few symptoms, and the benefits obtained in terms of complications during the procedure, the quality of life obtained and further ischemic events avoided should be evaluated systematically.

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