

Dose All of the Coronary Fistulas been Created by the Reopening Wire After Successful Revascularization of Chronic Total Occlusion?

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Abstract

Background: Coronary fistulas may be congenital or acquired generally as consequence of coronary interventions, mainly chronic total occlusion (CTO) reopening. When the reopening wire passes through the occlusion it may microperforate the advential vascular layers, favoring the fistulous communication between coronary vessel and cardiac chambers. But some of acquired coronary fistulas (ACFs) had been already present at the CTO vessels and would be seen after revascularization. This study was designed to investigate the characteristics of ACFs, which albeit mostly benign can cause concern and unnecessary treatment post successful CTO percutaneous coronary intervention (PCI).

Methods: Data, including clinical and procedural characteristics, medical history, and findings in electrocardiography, echocardiography and coronary angiography, from 2169 consecutive patients undergoing CTO PCI between January 2018 and December 2019 were analyzed retrospectively.

Results: 1844 (85.0%) underwent successful CTO PCI with complete revascularization. Among them, there were 49 cases (mean age, 62.80 ± 11.24 years; 40 men) of ACFs: 24 (49%) involved the right coronary artery, 19 (38.8%) the left anterior descending artery, and 6 (12.2%) the circumflex branch; and 38 (77.6%) were coupled with multiple fistulas (≥ 3), and 29 (59.2%) affected multiple branches of the CTO vessel (≥ 3). The majority of patients with ACFs had a history of MI or Q-wave ($n=34$, 69.4%), and angina was the most common complaint ($n=41$, 83.7%). None of them had pericardial effusion, tamponade and Hemodynamic abnormalities before or after PCI.

Conclusion: ACFs after successful CTO PCI mostly developed in patients with MI history, originated from the right coronary artery or left anterior descending artery, and involved multiple fistulas and CTO vessel branches.

Introduction

Coronary fistulas first described as congenital and abnormal vascular connections between coronary arteries and cardiac chambers or with other vessels, were later found to have other etiologies[1], including trauma, surgery, severe coronary atherosclerosis and myocardial infarction (MI). As first reported by Ryan in 1977[2] and later by others[3–5], MI is a common cause of acquired coronary fistulas (ACFs). ACFs secondary to MI are harmless, common in patients with chronic total occlusion (CTO), and hard to find before complete revascularization because of insufficient collateral filling. With higher rates of successful percutaneous coronary intervention (PCI) of CTO with new equipment and techniques, more ACFs are visualized after CTO PCI. It is important to distinguish ACF from coronary perforation (CP), a rare but potentially serious complication in PCI which can lead to pericardial effusion and tamponade, often necessitating medical treatment and even emergency pericardiocentesis or cardiac surgery[6]. To this end, there has not been a systematic description of imaging characteristics, medical history, and predictors of ACFs in a real-world cohort of patients who underwent successful CTO revascularization, which is therefore the subject of this study.

Methods

Patients

We retrospectively analyzed 2169 consecutive patients who underwent CTO PCI at ZhongShan Hospital of Fudan University from January 2018 to December 2019. Patients were excluded if the PCI was undertaken in the setting of an acute coronary syndrome. All original angiograms were reviewed by two physicians to confirm ACFs and to extract other clinical information. The study was approved by the institutional review board and all patients signed a general informed consent form.

Procedure

All patients were treated with oral dual antiplatelet drug before the procedure, unfractionated heparin was administered intravenously at 100 IU/Kg followed by further heparin as necessary to achieve a target activated clotting time of 250–350 s. Microcatheter include Finecross (Terumo, Japan) and Corsair (Asahi, Japan), and CTO guidewire include Fielder XT (Asahi, Japan), Fielder XTR (Asahi, Japan), Gaia 2 and 3 (Asahi, Japan), Pilot 150 and 200 (Abbott, America) had been used widely during the procedure.

Study definitions

CTO was defined as a complete occlusion with thrombolysis in myocardial infarction (TIMI) flow grade 0 antegrade for ≥ 3 months[7]. Procedural success was defined as $< 50\%$ residual stenosis with antegrade TIMI flow grade 3 at the end of the procedure[8]. ACF was defined as an abnormal connection between coronary arteries and cardiac chambers or with other vessels, which are secondary to exogenous or endogenous injury[1]. History of myocardial infarction was identified by past medical record including ST-segment elevation myocardial infarction (STEMI) or non-STEMI (NSTEMI) based on initial ECG, and clinical and laboratory findings. Adverse events captured included death from all causes, stent thrombosis (ST)/Q-wave myocardial infarction, emergent cardiac surgery and cardiac tamponade. Degree of development of coronary collaterals in CTO was classified using Rentrop Grade as follows[9]: 0 = none; 1 = filling of side branches of the artery to be dilated via collateral channels without visualization of the epicardial segment; 2 = partial epicardial filling of the occluded artery; and 3 = complete epicardial filling of the occluded artery.

Data collection

Demographic, procedural, and medical data were obtained from review of the catheterization laboratory database and medical records of patients.

Statistical analysis

Continuous variables are presented as mean \pm SD and were compared using t-test. Categorical variables are presented as counts and percentages and were compared using X^2 test or Fisher's exact test. All analyses were performed using SPSS V26, and $p < 0.05$ was considered statistically significant.

Results

Baseline clinical characteristics

Among the 2169 consecutive patients who underwent CTO PCI, procedural success was achieved in 1844 (85.0%). Among these 1844 patients, ACFs were found at the end of the procedure in 49 cases with mean age of 62.80 ± 11.24 years; 40 men (81.6%); 25 (51.0%) hypertensives, 15 (30.6%) diabetics, 5 (10.2%) with hyperlipidemia; 23 long-term smokers; and 10 with previous PCI and none with previous coronary artery bypass surgery (CABG); microcatheter and guidewire which had been used to cross the occlusion are listed in Table 1.

Table 1
Demographic and baseline clinical characteristics of patients with ACFs

Total patients with ACFs	N = 49
Age (years)	62.8 ± 11.24
Male	40 (81.60)
Symptom	
Asymptomatic	4 (8.2)
Stable angina	24 (49.0)
Unstable angina	17 (34.7)
Dyspnea	2 (4.1)
Syncope	2 (4.1)
New York Heart Association (NYHA) class	
□	7 (14.3)
□	26 (53.1)
□	11 (22.4)
□	5 (10.2)
Hypertension	25 (51.0)
Diabetes	15 (30.6)
Hyperlipidemia	5 (10.2)
Smoking	23 (46.9)
History of MI	14 (28.6)
History of MI or Q-wave MI	34 (69.4)
Previous PCI	10 (20.4)
Previous CABG	0
Echocardiography	
LVPWT (mm)	9.63 ± 1.36
IVST (mm)	10.26 ± 1.88
LVEDD (mm)	41.47 ± 8.25
LVESD (mm)	54.14 ± 6.69
LVEF (%)	48.94 ± 9.76

Total patients with ACFs	N = 49
Ventricular Aneurysm	2 (4.1)
CTO technique	
Antegrade	44 (89.8)
Retrograde	5 (10.2)
Microcatheter	
Finecross	17(34.7)
Corsair	25(50.0)
Guidewire for crossing occlusions	
Fielder XTR	14(28.6)
GAIA 2	20(40.8)
GAIA 3	5(10.2)
Pilot 200	10(20.4)
Data are presented as n (%), or mean standard \pm deviation	
ACFs = acquired coronary fistulas; NYHA = New York Heart Association; MI = myocardial infarction; PCI = percutaneous coronary intervention; CABG = coronary artery bypass surgery; LVPWT = left ventricular posterior wall thickness; IVST = interventricular septum thickness; LVEDD = left ventricular end diastolic diameter; LVESD = Left Ventricular End Systolic Diameter; LVEF = left ventricular ejection fraction; CTO = chronic total occlusion.	

Clinical symptoms

The most common presenting complaint was angina (41, 83.7%), followed by asymptomatic (4, 8.2%), dyspnea (2, 4.1%) and syncope (2, 4.1%). By NYHA classification, 7 patients were class I, 26 class II, 11 class III and 5 class IV.

Electrocardiogram (ECG) and Echocardiography

As shown in Fig. 1, among the 49 cases with ACFs, ECG displayed Q waves in 31 (63.3%), was normal in 10 (20.4%), and had ST-T changes (depressed ST segment; low and flat T wave) in 8 (16.3%); 47 cases maintained sinus rhythm, and 2 had atrial fibrillation (AF).

Echocardiography examination was performed in all of patients. Mean left ventricular ejection fraction (LVEF) was $48.94 \pm 9.76\%$, $< 40\%$ in 10 cases, $40\%-49\%$ in 14, and $\geq 50\%$ in 25 cases. Forty-two patients

(85.7%) had left ventricular systolic dysfunction (LVSD), and 2 ventricular aneurysm. Mean left ventricular posterior wall thickness (LVPWT), interventricular septum thickness (IVST), left ventricular end diastolic diameter (LVEDD) and left ventricular end systolic diameter (LVESD) are shown in Table 1. None pericardial effusion and tamponade had been found in patients with ACFs.

Coronary angiography

Table 2 presents the angiographic characteristics of ACFs. The most frequent origin of the ACFs was the right coronary artery (RCA; n = 24; 49.0%) followed by the left anterior descending artery (LAD; n = 19; 38.8%) and the left circumflex coronary artery (LCX; n = 6; 12.2%), with none originating in the distal left main artery and 11 (22.4%) found during angiography through collateral vessels; 38 (77.6%) were associated with multiple fistulas (≥ 3), and 29 (59.2%) involved multiple branches of CTO vessel (≥ 3). Images of ACFs are shown in Fig. 2 and Movie S1-S6 in Supplementary.

Table 2
Angiographic characteristics of ACFs in CTO

Total patients with ACFs	n = 49
Right coronary artery dominance	45 (91.8)
CTO vessel	
LAD	19 (38.8)
LCX	6 (12.2)
RCA	24 (49.0)
Number of ACFs holes	
<3	11 (22.4)
≥3	38 (77.6)
Number of affected vessel branches	
< 3	20 (40.8)
≥3	29 (59.2)
Collateral filling of Rentrop	
0	0 (0)
1	28 (57.1)
2	17 (34.7)
3	4 (8.2)
Diagnosed by collateral circulation before PCI	11 (22.4)
Data are presented as n (%)	
LAD = left anterior descending; LCX = left circumflex coronary artery; RCA = right coronary artery; CAG = coronary angiography.	

As shown in Table 3, clinical and imaging characteristics and medical history parameters were similarly distributed among cases of ACF originating from RCA, LAD and LCX, except for smoking that was more frequent in cases of ACFs originating in RCA and LAD than LCX. The mean length of hospital stay was 4.45 days, and there were no in-hospital cases of death, emergent cardiac surgery or MI.

Table 3
Comparison of clinical and angiographic characteristics among the three CTO vessels subgroups

	LAD (n = 19)	LCX (n = 6)	RCA (n = 24)	P value
Age (years)	62.21 ± 9.60	63.83 ± 12.06	63.00 ± 12.62	0.948
Male	17 (89.47)	3 (50.00)	20 (83.33)	0.089
Smoking	9 (47.37)	0	14 (58.33)	0.038*
Hypertension	7 (36.84)	4 (66.67)	14 (58.33)	0.268
Diabetes	6 (31.58)	2 (33.33)	7 (29.17)	0.974
Hyperlipidemia	2 (10.53)	0	3 (12.50)	0.663
Previous PCI	2 (10.53)	1 (16.67)	7 (29.17)	0.312
With Clinical symptoms	16 (84.21)	6 (100)	23 (95.83)	0.284
History of MI	4 (21.05)	3 (50.00)	7 (29.17)	0.391
HYNA \bar{x} - \bar{x}	4 (21.05)	3 (50.00)	9 (37.50)	0.326
Q-wave	13 (68.42)	4 (66.67)	14 (58.33)	0.779
LVEF (%)	48.63 ± 11.97	43.83 ± 11.04	50.46 ± 7.14	0.333
≥ 3 holes	14 (73.68)	3 (50.00)	21 (87.50)	0.126
≥ 3 branches vessel	11 (57.89)	3 (50.00)	15 (62.50)	0.847
Hospital stay (days)	4.79 ± 2.42	4.5 ± 1.6	4.17 ± 1.71	0.603
Data are presented as n (%), or mean standard ± deviation				

Discussion

In the present study, the most common characteristics of ACFs in successful CTO PCI patients included: multiplicity and diffusibility with three or more branch vessels and drainage sites; and a history of MI or Q-Wave (69.4% vs. 56.3%-58.4% in the overall CTO population)[10, 11]. Smoking is a risk factor for coronary artery disease (CHD), and is more frequent among ACFs originating from LAD and RCA, which could be secondary to smaller sample size or lower incidence in LCX. In the presence of ACFs, the contrast agent quickly diffuses out of the coronary artery without retention during angiography.

In our data, the prevalence of ACFs in successful CTO PCI patients was 2.60%, which is lower than that the up to 8.9% one for CP during CTO PCI[12]. On occasions, CP and ACFs have similar angiographic

appearance, and Ellis classification has been used to classify CP as type I–III[13]. Unlike CP, ACFs drain into the cardiac chambers, and therefore pericardial effusion and tamponade do not develop thereby rendering procedures safe. Wrong judgment of operator and lack of bedside echocardiography may lead to concern and unnecessary treatment, such as coil embolization which would be feasible and effective in treating CP, but useless and unnecessary in ACFs (Fig. 3, Movie S7 and S8 in Supplementary). It is therefore important to distinguish ACFs from CP during CTO PCI.

ACFs could result from MI, hypertrophic cardiomyopathy, dilated cardiomyopathy, tumor, PCI, CABG, cardiac transplant, cardiac biopsy, and pacemaker placement, among other etiologies[14–16]. Rupture of the microvasculature may be the most important cause of ACFs[17, 18]. A sudden total occlusion of a coronary artery blocks blood flow to the heart muscle leading to tissue death. Because of extensive myocardial cell necrosis, myocardial rupture will occur within the first two weeks in 90% of MI[19, 20]. Complete rupture triggers hemopericardium and sudden death, and incomplete rupture will become a ventricular aneurysm because the pericardium seals the ventricular perforation. In CTO patients, in contrast, the coronary artery obturates slowly allowing development of collateral circulation, and myocardial death does not cause symptoms and patients can survive the attack, but death of endocardium damages the microvasculature. Myocardial scarring and rupture of the microvasculature causes ACFs, which become apparent by coronary angiography after successful CTO-PCI. In some of the patients with ACFs and good collaterals and no history of MI or Q-Wave, other underlying mechanisms for ACFs include newly developed collaterals, neo-vascularization of mural thrombus formation and reopening of the thebesian vessels[21].

ACFs caused by MI drain into LV more than right ventricle (RV)[5, 22], possibly because: (a) right ventricular myocardial infarction may not result in significant ischemia or infarction; the thin walls of the RV require less oxygen, and the ability of RV to extract oxygen is increased during MI; rich collaterals from LAD reduce infarct size, and the RV has direct blood supply from the RV cavity through the thebesian veins, with coronary perfusion of the RV occurring both in systole and diastole[23, 24]; and (b) most of the ACFs are located at the end of the coronary arteries which mainly supply the LV. Moreover, no coronary steal syndrome or hemodynamic impairment was apparent after CTO PCI in our patients with ACFs.

Limitations

Because the drainage sites were tiny, we couldn't clear and confirm where the drainage sites of ACFs located by Transthoracic Echocardiogram (TTE), but no pericardial effusion and tamponade had been confirm after PCI. ACFs have been reported to cause myocardial ischemia, endocarditis and congestive heart failure[25], but long-term tracking are needed to evaluate whether ACFs are associated with poor prognosis in CTO patients.

Conclusion

In the present study, It is very important to recognize ACFs, which is difficult to distinguish with CP during CTO PCI. ACFs albeit mostly benign can cause concern and unnecessary treatment, such as coil embolization. The characteristic of ACFs are diffusibility, asymptomatic, distal coronary artery-left ventricular fistulas in patients with history of MI.

Abbreviations

ACFs: acquired coronary fistulas; CTO: chronic total occlusion; PCI: percutaneous coronary intervention; CP: coronary perforation; LVEF: left ventricular ejection fraction; LVSD: Left ventricular systolic dysfunction.

Declarations

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Competing interests

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

Authors' contributions

FR drafted the manuscript. TH, SY and YY did collect data and statistical analysis. YW and WF reviewed All original angiograms, LH and FC provides the figures and their interpretation, FM, HZ and GJ helped revising the manuscript for important intellectual content. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

All patients signed written informed consent form. Fudan University affiliated Zhongshan Hospital Ethics Committee had approved the work and received all the written informed consent.

Consent for publication

Not applicable.

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Figures

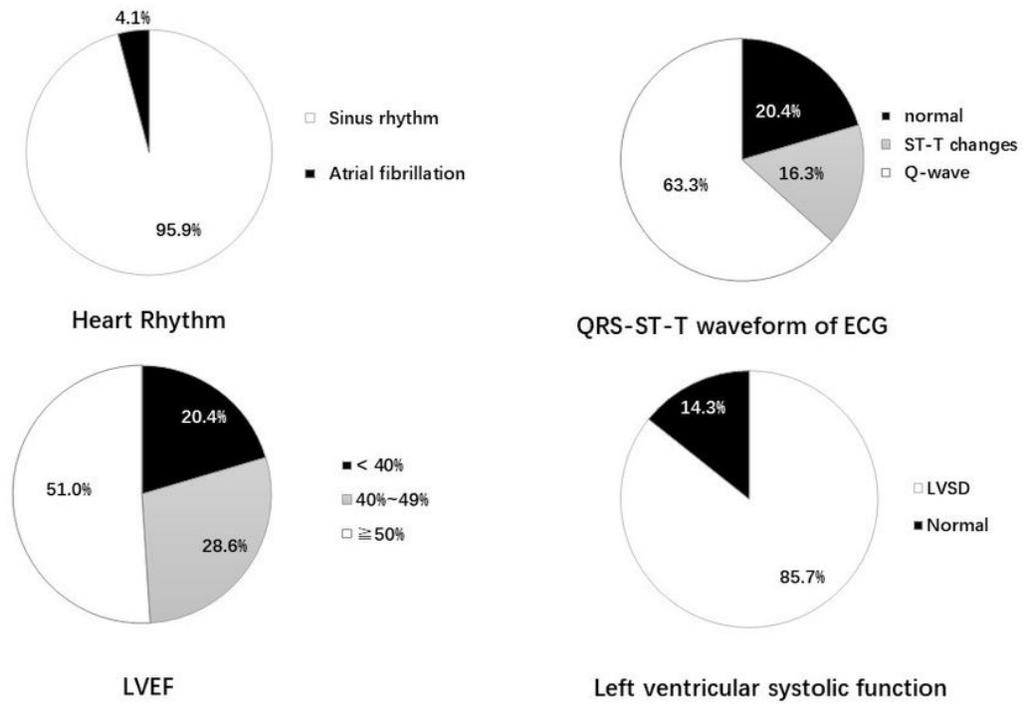


Figure 1

The statistical pie diagram of Heart Rhythm, QRT-ST-T waveform of ECG, LVEF and Left ventricular systolic function. ECG: electrocardiogram; ST-T change include depressed ST segment, Low and flat T wave; LVEF: left ventricular ejection fraction; LVSD: Left ventricular systolic dysfunction.

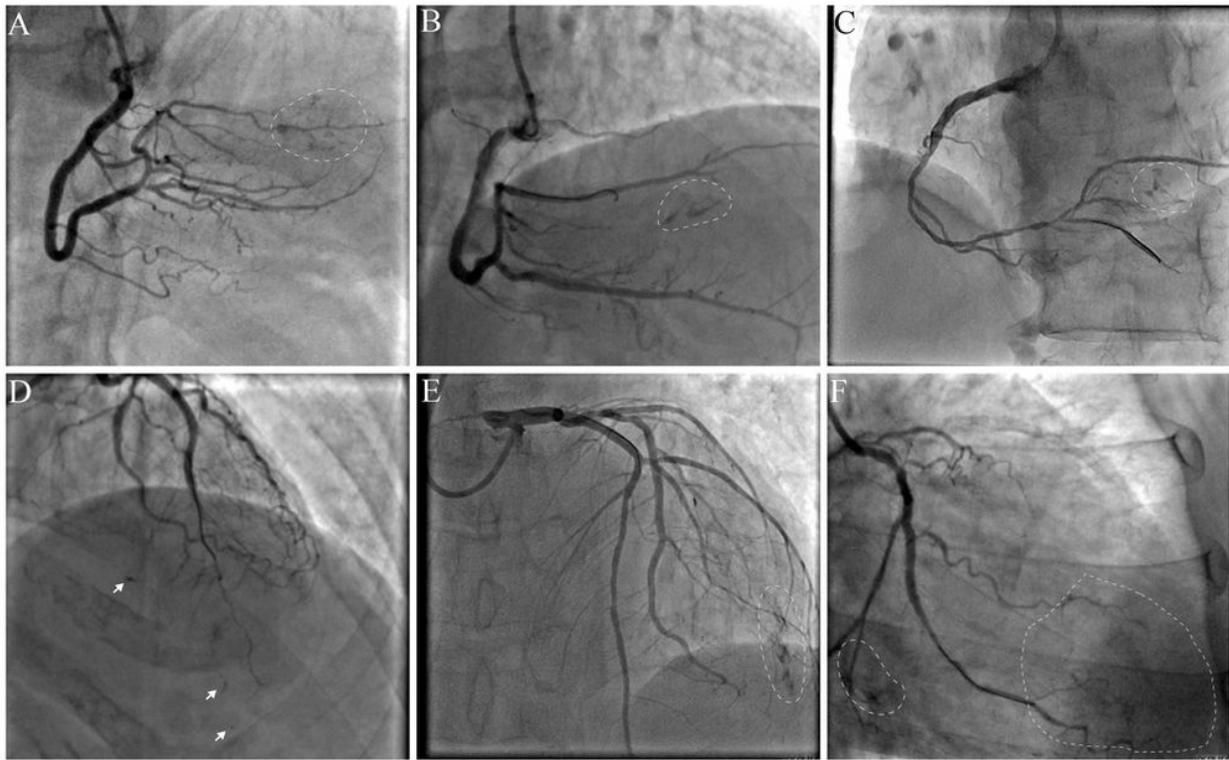


Figure 2

The angiographic characteristics of ACFs were Multiple, Diffuse, and Tiny. (A, B and C) ACFs were found after RCA CTO-PCI; (D and E) were found after LAD CTO-PCI; (F) was found after LCX CTO-PCI. (spot fistulas (arrow); flake fistulas (circle); Movie S1-S6 show angiographic finding and are included in Supplementary)

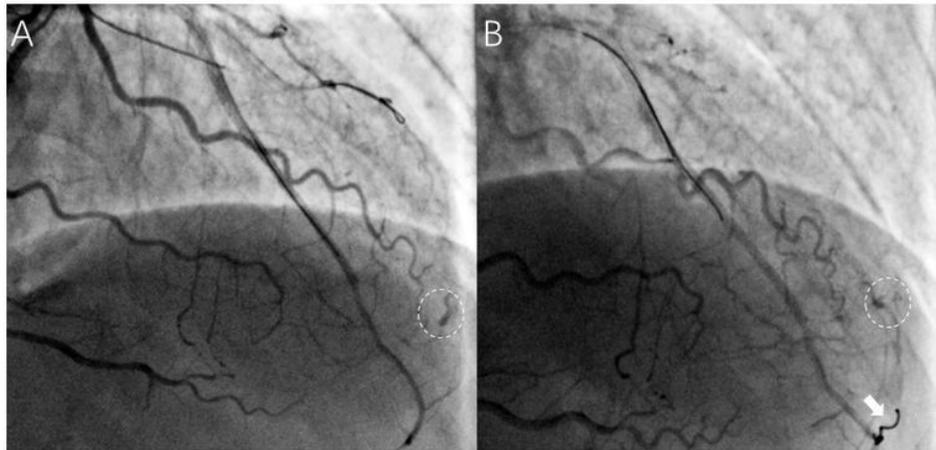


Figure 3

A case illustration of ACFs uselessly and unnecessarily treated with coil embolization. (A) After successful recanalization of the LAD, ACFs were clearly visible and difficult to identify with coronary perforation timely (see circle); (B) Rattled and successful delivery of one coil through the microcatheter (Finecross, Terumo, Japan) (see arrows), but ACFs still existed (see circle), and no pericardial effusion had been found by echocardiography. (movie S7-S8 show angiographic finding and are included in Supplementary)

Supplementary Files

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