

Case Report

## Relentless Angina of a Scarred Heart

Eslam Abbas<sup>1,\*</sup>, Ahmed Mahdy<sup>2,3</sup>, Shady Mansy<sup>3</sup>

<sup>1</sup>Department of Cardiology, Arab Contractors Medical Center (ACMC), Cairo, Egypt

<sup>2</sup>Department of Cardiology, Jaber Al-Ahmad Armed Forces Hospital, Kuwait City, Kuwait

<sup>3</sup>Department of Cardiology, Al Mokattam Insurance Hospital, Cairo, Egypt

### Abstract

Refractory anginal pain affects nearly 5-10% of stable coronary artery disease patients, and maximizing the anti-ischemic medical therapy is the standard first-line treatment. The presence of a scarred myocardial territory of the epicardial coronary chronic total occlusion (CTO) limits the implementation of other modalities, such as angioplasty and surgical bypass. Accordingly, this subset of patients, who show poor response to medical treatment with the absence of considerable reversible ischemia, bears an additional burden of persistent angina besides the structural and functional complications resulting from their scarred hearts. In this report, a patient, with compensated ischemic cardiomyopathy, complaining of disabling stable angina was indicated for diagnostic coronary angiography that showed a chronic total occlusion (CTO) at the mid-segment of the left anterior descending coronary artery (LAD) and otherwise no significant stenoses in the epicardial coronary tree. After the failure of maximized anti-ischemic medical therapy, the patient underwent elective percutaneous intervention (PCI) to the left anterior descending coronary artery (LAD) chronic total occlusion (CTO) with 2 overlapping drug-eluting stents that yielded a favorable outcome on patient follow-up even though a myocardial perfusion imaging failed to show considerable reversible ischemia at the left anterior descending coronary artery (LAD) territory. The report points out that elective chronic total occlusion (CTO) revascularization may alleviate anginal pain, despite the absence of a considerable macroscopic ischemia, after failure of a maximized anti-ischemic medical regimen.

### Keywords

Chest Pain, Refractory Angina, Elective PCI, CTO, Revascularization, Myocardial Ischemia

## 1. Case Presentation

A male patient, aged 65 years and known to have ischemic heart disease, was presented to the outpatient clinic with stable angina (CCS angina score III), that was not responding to anti-ischemic medical therapy. Physical examination was unremarkable except for mild lower limb edema. Evident risk factors were heavy smoking and a positive family history of CAD. The patient doesn't suffer

from diabetes or hypertension, besides there is no history of peripheral or cerebral vascular diseases. The patient is known to have compensated ischemic cardiomyopathy on anti-ischemic and anti-failure medications. He had undergone a PCI in 2014 wherein a balloon dilatation was performed followed by the deployment of a DES to the LAD for critical stenosis.

\*Corresponding author: [islam.omr@med.au.edu.eg](mailto:islam.omr@med.au.edu.eg) (Eslam Abbas)

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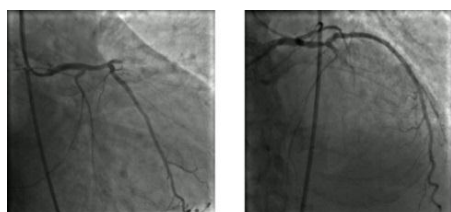
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## 2. Investigations

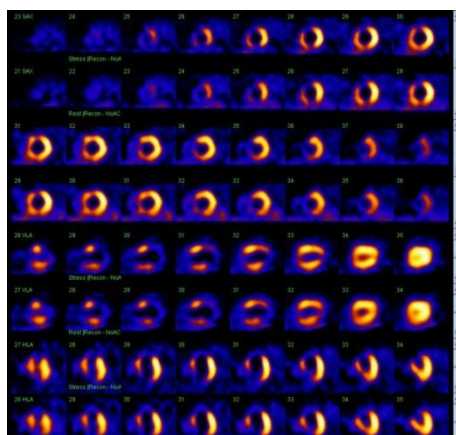
Laboratory investigations showed normal chemistry panel and electrolytes ranges, while CBC showed normal hemoglobin levels with mild leukocytosis. Baseline ECG showed pathological Q waves at V1-V4 and T wave inversion at V5, V6, I, and aVL. Baseline Echo showed no valvular abnormalities, dilated left ventricle with apical akinesia, and impaired LV systolic function with an estimated EF of 37%, while low dose dobutamine echo showed non-viable LAD territory.

## 3. Management

The patient was indicated for diagnostic CA to address the flow state in the coronary circulation and detect stenotic lesions that provoke the patient's anginal pain. The angiography showed a mid-LAD instent CTO (*Figure 1*), and otherwise no significant stenoses within the epicardial coronary circulation. A Tc-99m Sesta-MIBI SPECT was done and showed a fixed perfusion defect of scarred tissue at the apex, antero-apical, infero-apical, mid-anterior, and antero-septal segments (*Figure 2*) with a total defect size of 30%. A cardiovascular magnetic resonance (CMR) with late gadolinium enhancement imaging, which has a higher specificity and lesser sensitivity [1], was not feasible due to an orthopedic implant.



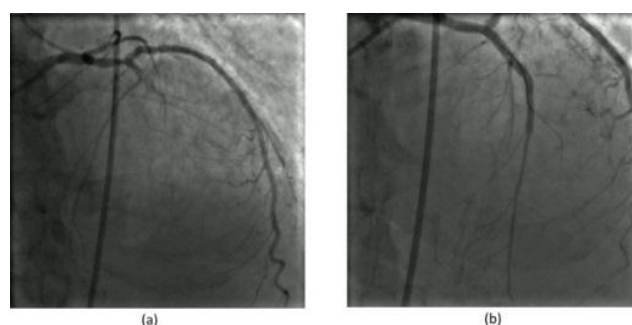
**Figure 1.** Two pre-stenting angiograms showing the mid-LAD instent CTO.



**Figure 2.** A SPECT MPI (splash view) showing a positive test with a fixed perfusion defect of scarred tissue at the apex, antero-apical, infero-apical, mid-anterior, and antero-septal segments.

Accordingly, and as the patient's anginal pain is unresponsive to standard anti-ischemic medications; the patient was put on a maximized anti-ischemic medical regimen in an attempt to neutralize a suggested role of a diseased coronary microvasculature. The prescribed regimen included atorvastatin, aspirin, high dose  $\beta$ -blocker, ivabradine, nicorandil, and trimetazidine with an optimum heart rate ranging between 55-60 bpm during follow-up [2]. The patient was already on sacubitril-valsartan as an anti-failure medication. Calcium channel blockers were not warranted due to low EF and to avoid patient decompensation [3]. Follow-up laboratory tests were within normal values.

After 3 months of regular follow-up, the patient was still complaining of typical chest pain that has not shown significant improvement after the maximization of anti-ischemic treatments. Consequently; the heart team decided to proceed for PCI to LAD instent CTO (*Figure 3*). The procedure was performed using the antegrade wire escalation (AWE) technique [4, 5] then two overlapping DESs (Resolute Onyx  $2.5 \times 18$  inflated at 20 atm and Ultimaster  $2.5 \times 33$  inflated at 14 atm from distal to proximal) were deployed.



**Figure 3.** Two angiograms before (a) and after (b) elective PCI to the mid-LAD CTO.

## 4. Follow-Up

The patient showed immediate satisfactory anginal relief. The previously mentioned maximized anti-ischemic medical regimen was reduced to ticagrelor, bisoprolol, atorvastatin, and sublingual dinitrate on-demand with maintained sacubitril-valsartan as anti-failure medication. However, the fading of the activity-limiting angina unmasked manifestations of heart failure which have surfaced in the form of dyspnea (NYHA score II). Still, the patient's ability to perform routine activities and thus the quality of life was significantly improved as assessed by the Seattle Angina Questionnaire [6]. Additionally, the patient's immediate relief of anginal pain was subjected to a long-term period of regular follow-up in an attempt to exclude a suggested role of the placebo effect.

## 5. Discussion

Chronic total occlusion has usually been neglected as a target for revascularization with elective PCI due to technical complexity, procedure length, and increased failure rates [7]. However, two randomized controlled clinical trials [8, 9] and several observational studies [10] have shown that PCI to CTO leads to a marked improvement in the quality of life and symptoms of patients who suffer a CTO-induced ischemic burden. In an ischemia-based algorithm, patients with normal wall motion or hypokinesia are indicated for revascularization if the ischemic myocardium is more than 10% of the left ventricle [11]. Meanwhile, patients with akinesia or dyskinesia are indicated for revascularization only if a viability assessment shows a viable myocardium [12].

In the presented case, SPECT imaging showed a scarred myocardium within the territory of the mid-LAD-CTO without considerable ischemia, so maximizing the anti-ischemic medical therapy was the standard treatment option. The patient was informed of the limitations of additional therapeutic options for his case after the failure of the maximized medical regimen, and revascularization (or bypassing surgical intervention) of his LAD was not recommended due to the non-viability of the vascular territory and may result in risky complications [10, 13]. Yet; the patient endorsed the interventional procedure.

The rationale of the interventional reperfusion therapy in the presented case aimed to achieve a decisive modulation of pathologies in the cardiac microenvironment using interventions to the microvasculature of the epicardial coronaries [14] after the failure of medical therapy. The target modulation was relieving a Tc-99m Sesta-MIBI SPECT undetected ischemic burden which may affect territories of the inferior wall if the occluded LAD is a big wrap-around vessel, or the myocytes at the interface between the scarred and the healthy myocardial tissue. Well-developed collateral circulation and preconditioning are not sufficient to substantially reduce ischemia [15] and can lead to poor demarcation between the scar and the healthy myocardium, wherein the interface territory shows a hibernated viability and suffers chronic ischemia that leads to anginal pain.

## 6. Conclusion

In an ischemic-based algorithm, patients without a considerable macroscopic ischemic burden (less than 10% of the left ventricle) may benefit from elective revascularization of a chronic total occlusion to alleviate anginal pain after the failure of a maximized anti-ischemic medical regimen.

## Abbreviations

CAD	Coronary Artery Disease
PCI	Percutaneous Intervention

CTO	Chronic Total Occlusion
DES	Drug-Eluting Stent
LAD	Left Anterior Descending Artery
SPECT-MPI	Single-Photon Emission Computed Tomography, Myocardial Perfusion Imaging

## Author Contributions

AM and SM performed the intervention. EA performed the investigations and wrote the manuscript with support from AM and SM. All authors contributed equally to this work, and all authors have read and approved the manuscript.

## Ethics Approval and Consent to Participate

The authors confirm they comply with human studies committees' regulations of the authors' institutions and COPE guidelines, including patient consent where appropriate.

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## Data Availability and Sharing

Data can be shared with researchers who submit a proposal with a valuable research question. Requests should be directed to the corresponding author.

## Conflict of Interest

The authors declare that they have no competing interests. The article was prepared in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

## References

- [1] Emrich T, Halfmann M, Schoepf UJ, Kreitner K-F. CMR for myocardial characterization in ischemic heart disease: state-of-the-art and future developments. *Eur Radiol Exp*. 2021; 5(1): 1–13.
- [2] Davies A, Fox K, Galassi AR, Banai S, Ylä-Herttuala S, Lüscher TF. Management of refractory angina: an update. *Eur Heart J*. 2021; 42(3): 269–83.
- [3] Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE, Drazner MH, Fonarow GC, Geraci SA, Horwich T, Januzzi JL. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2013; 62(16): e147–239.

- [4] Berkhout T, Claessen BE, Dirksen MT. Advances in percutaneous coronary intervention for chronic total occlusions: current antegrade dissection and reentry techniques and updated algorithm. *Netherlands Hear J*. 2021; 29(1): 52–9.
- [5] Maeremans J, Knaapen P, Stuijzand WJ, Kayaert P, Pereira B, Barbato E, Dens J. Antegrade wire escalation for chronic total occlusions in coronary arteries: simple algorithms as a key to success. *J Cardiovasc Med*. 2016; 17(9): 680–6.
- [6] Thomas M, Jones PG, Arnold S V, Spertus JA. Interpretation of the Seattle Angina Questionnaire as an Outcome Measure in Clinical Trials and Clinical Care A Review. *JAMA Cardiol*. 2021; 6(5): 593–9.
- [7] Bardaj í A, Rodríguez-López J, Torres-Sánchez M. Chronic total occlusion: to treat or not to treat. *World J Cardiol*. 2014; 6(7): 621.
- [8] Werner GS, Martin-Yuste V, Hildick-Smith D, Boudou N, Sianos G, Gelev V, Rumoroso JR, Erglis A, Christiansen EH, Escaned J. A randomized multicentre trial to compare revascularization with optimal medical therapy for the treatment of chronic total coronary occlusions. *Eur Heart J*. 2018; 39(26): 2484–93.
- [9] Obedinskiy AA, Kretoy EI, Boukhris M, Kurbatov VP, Osiev AG, Ibn Elhadj Z, Obedinskaya NR, Kasbaoui S, Grazhdankin IO, Prokhorikhin AA, et al. The IMPACTOR-CTO Trial. *JACC Cardiovasc Interv*. 2018 Jul; 11(13): 1309–11.
- [10] Sapontis J, Salisbury AC, Yeh RW, Cohen DJ, Hirai T, Lombardi W, McCabe JM, Karpaliotis D, Moses J, Nicholson WJ. Early procedural and health status outcomes after chronic total occlusion angioplasty: a report from the OPEN-CTO registry (Outcomes, Patient Health Status, and Efficiency in Chronic Total Occlusion Hybrid Procedures). *JACC Cardiovasc Interv*. 2017; 10(15): 1523–34.
- [11] Windecker S, Kolh P, Alfonso F, Collet J-P, Cremer J, Falk V, Filippatos G, Hamm C, Head SJ. 2014 ESC/EACTS Guidelines on myocardial revascularization. *Eur Heart J*. 2014 Oct 1; 35(37): 2541–619.
- [12] Schumacher SP, Stuijzand WJ, Opolski MP, van Rossum AC, Nap A, Knaapen P. Percutaneous coronary intervention of chronic total occlusions: when and how to treat. *Cardiovasc Revascularization Med*. 2019; 20(6): 513–22.
- [13] Rigger J, Hanratty CG, Walsh SJ. Common and Uncommon CTO Complications. *Interv Cardiol Rev*. 2018; 13(3): 121.
- [14] Myat A, Patel M, Silberbauer J, Hildick-Smith D. Chronic total coronary occlusion revascularisation positively modifies infarct-related myocardial scar responsible for recurrent ventricular tachycardia. *EuroIntervention J Eur Collab with Work Gr Interv Cardiol Eur Soc Cardiol*. 2019.
- [15] Sachdeva R, Agrawal M, Flynn SE, Werner GS, Uretsky BF. The myocardium supplied by a chronic total occlusion is a persistently ischemic zone. *Catheter Cardiovasc Interv*. 2014; 83(1): 9–16.