ESC European Society of Cardiology
European Heart Journal - Case Reports (2024) **8**, 1–3
https://doi.org/10.1093/ehjcr/ytae082

Coronary branch steal due to an anomalous single and diseased vessel

Alexandru Achim [1], Danai Kitkungvan[2], Richard L. Kirkeeide[2], and Nils P. Johnson [2]*

[1]Department of Cardiology, Nicolae Stăncioiu Heart Institute, Iuliu Haţieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania; and [2]Weatherhead PET Center, Division of Cardiology, Department of Medicine, McGovern Medical School at UTHealth and Memorial Hermann Hospital, 6431 Fannin St, Houston, TX 77030, USA

Received 19 September 2023; revised 17 December 2023; accepted 7 February 2024; online publish-ahead-of-print 12 February 2024

Coronary steal describes the end result of a variety of mechanisms that cause stress flow to fall below resting levels.[1] The most common scenario arises from a collateral-dependent chronic total occlusion (CTO) with a diseased donor vessel. While resting flow remains intact due to vasodilation of the CTO bed, pressure loss in the donor artery during hyperaemia leads to a drop in distal coronary pressure at the origin of the collaterals. Since flow follows pressure, collateral blood supply to the CTO decreases due to lower upstream pressure in the donor plus an inability of the CTO bed to vasodilate further. Hence, the paradox: pharmacologic 'hyper'-aemia produces 'hypo'-aemia, namely flow falling below baseline levels (fancifully termed 'steal', although collateral flow does not actually reverse direction).

However, coronary steal can arise from serial epicardial disease in a branched system without a frank CTO, so-called branch steal.[2] As a specific example, we present the case of a 68-year-old woman who developed severe angina during the course of her treatment for breast cancer. Invasive angiography demonstrated a single coronary artery originating from the right sinus, a so-called Lipton type R-I anomaly (*Figure 1A*; see Supplementary material online, *Video S1*).[3] A moderate lesion in the atrioventricular groove portion of the vessel between the posterior descending artery (PDA) and functional obtuse marginal branches underwent physiologic evaluation using positron emission tomography (PET) imaging. The small calibre left anterior descending artery (LAD) outflow demonstrated diffuse disease.

Stress uptake, coronary flow reserve (CFR), and coronary flow capacity (CFC)[4] maps quantified frank ischaemia in the proximal LAD distribution with CFR < 1 coronary steal in ∼20% of the left ventricle (LV; *Figure 2*). After successful imaging-guided percutaneous coronary

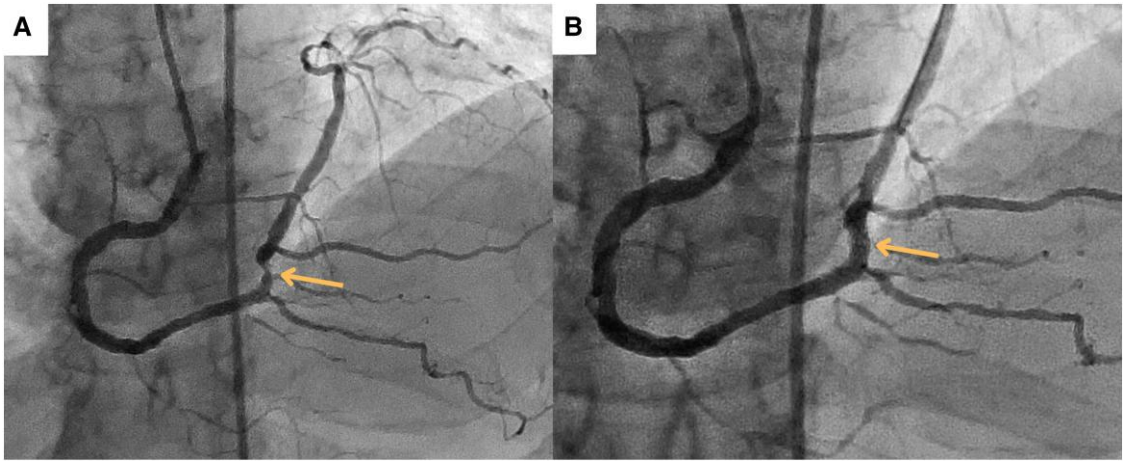

Figure 1 (*A*) Invasive coronary angiography demonstrated a single vessel originating from the right sinus and proceeding along the atrioventricular groove, culminating in the left anterior descending artery—a so-called Lipton type R-I anomaly.[3] (*B*) Repeat angiography after successful imaging-guided implantation of a 3.5 × 16 mm drug-eluting stent (the arrows indicate the lesion).

* Corresponding author. Tel: +1 713 500 6611, Email: Nils.Johnson@uth.tmc.edu
Handling Editor: Flemming Javier Olsen
Peer-reviewers: Milenko Zoran Cankovic and Sumit Sohal

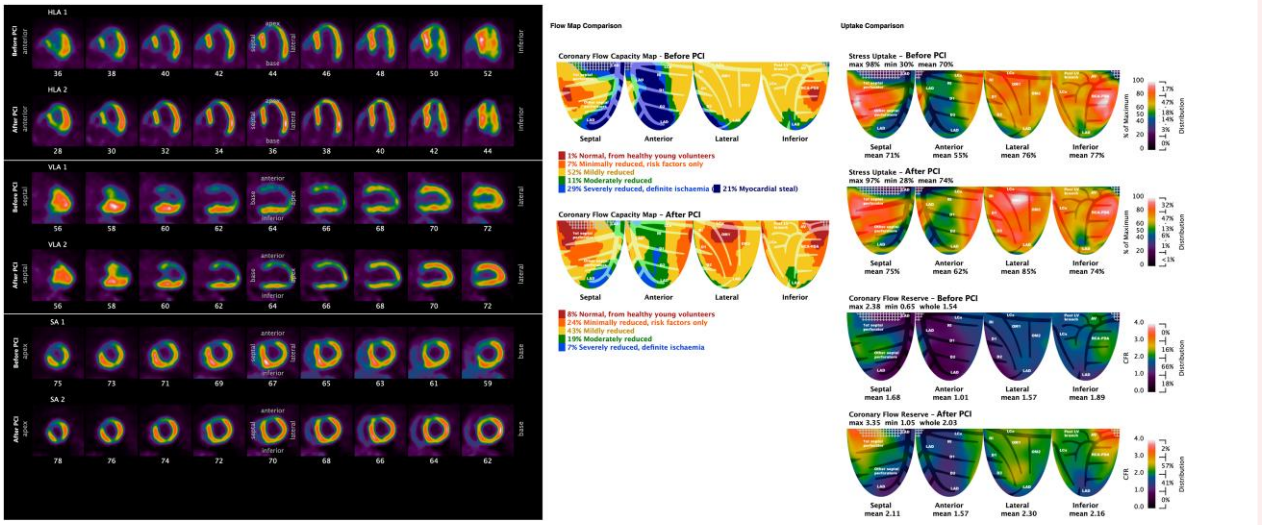

Figure 2 Positron emission tomography perfusion images display relative stress uptake (left portion) before (top row) and after (bottom row) intervention on the left anterior descending artery. Topographic ('surface') maps of coronary flow capacity (middle portion), stress uptake (top of right portion), and coronary flow reserve (bottom of right portion) pair before vs. after results.

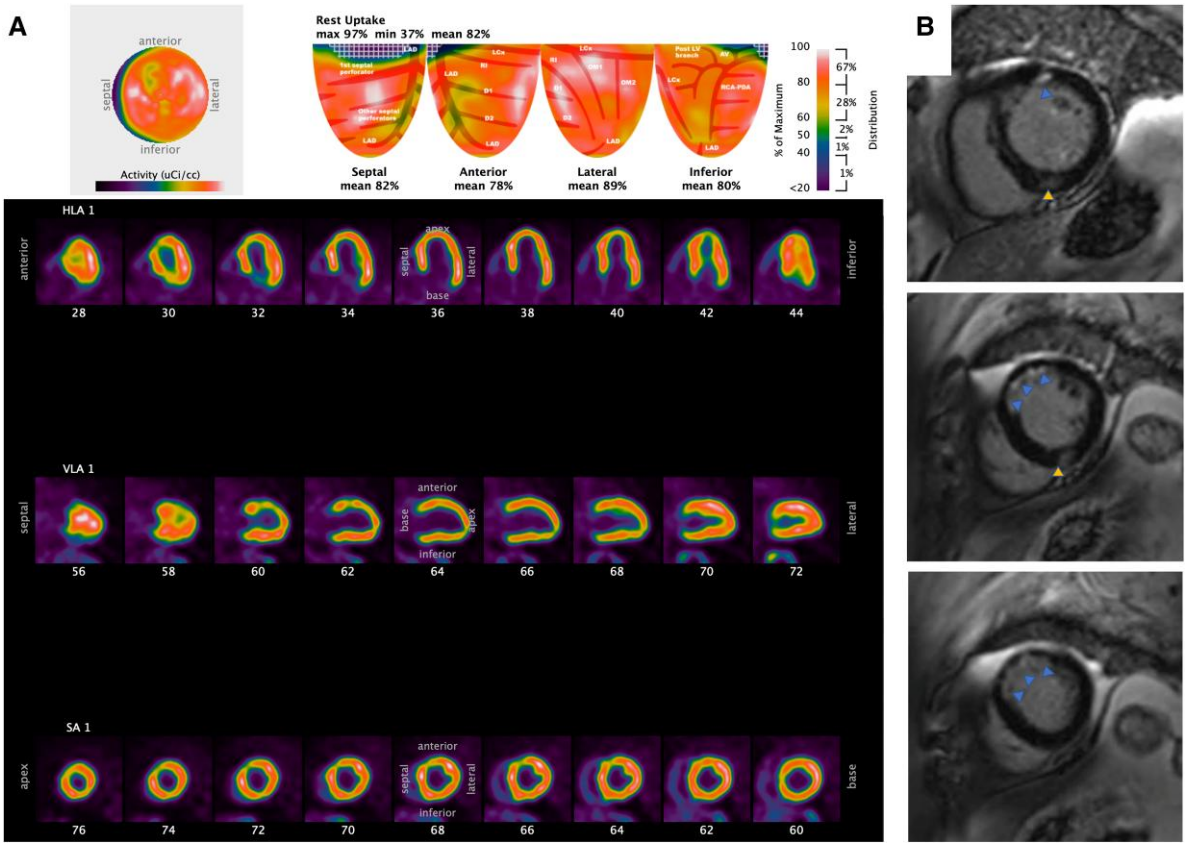

Figure 3 Resting positron emission tomography (*A*) and cardiac magnetic resonance images (*B*) of subendocardial scar; the blue arrows in (*B*) show coronary artery disease–related late gadolinium enhancement in the mid-left anterior descending artery distribution (7% left ventricular burden), and the yellow arrows depict non-coronary artery disease–related enhancement at the right ventricular insertion site (2% left ventricular burden).

intervention (PCI) of the mid-vessel with a 3.5 × 16 mm drug-eluting stent (*Figure 1B*), PET imaging was repeated 1 month later (*Figure 2*). Coronary steal had now resolved since 0% of the LV displayed CFR < 1, although a smaller and less intense relative uptake defect persisted. Average CFR in the entire anterior quadrant increased from 1.01 to 1.57—a >50% improvement consistent with successful revascularization. The amount of moderately to severely reduced (green or blue) CFC that accounts for varying resting perfusion decreased from 40 to 26% of the LV. The LAD quadrant experienced the greatest benefit from PCI (the lateral and inferior territories with a 46 and 14% CFR improvement, respectively), underscoring the positive impact of revascularization in a single conduit-like coronary system.

Branch steal and its resolution after PCI in this case can be understood as follows: At rest, the LAD territory vasodilates to maintain its blood flow despite combined viscous pressure loss along the entire course of the single coronary artery plus separation loss at the focal stenosis. During hyperaemia, viscous pressure loss increases almost two-fold (average CFR in the inferior quadrant before PCI was 1.89) with a superimposed, roughly quadrupled separation loss. By the LAD origin, both hyperaemic pressure and flow have fallen below baseline ('hypo'-emia) due to augmented flow ('hyper'-emia) in the upstream PDA and obtuse marginal territories (sequential average quadrant CFR 1.89 to 1.57 to 1.01 from inferior to lateral to anterior). Percutaneous coronary intervention eliminated the separation pressure loss but left the substantial viscous pressure loss along the single conduit plus diffuse outflow LAD disease. Repetitive ischaemic episodes and/or non-occlusive plaque rupture in the mid-LAD produced a small (<10% LV), non-transmural scar, as evidenced by a mild resting PET anterior uptake defect as well as subendocardial delayed hyperenhancement without acute oedema via cardiac magnetic resonance imaging (*Figure 3*).

To our knowledge, this case represents the first quantified example of branch steal due to an anomalous single coronary artery and its resolution after successful PCI.

Lead author biography

Dr Alexandru Achim is an interventional cardiologist originating from Cluj-Napoca, Romania, with a fervent passion for coronary and structural interventions. He pursued his residency training in prestigious medical institutions, including Graz (Austria), Basel (Switzerland), and Szeged (Hungary). It was at the University of Szeged where he completed his PhD studies in 2023. His research contributions have primarily centred around vascular access, coronary physiology, and CTO PCI. Currently, Dr Achim serves as a cardiology specialist in Cluj-Napoca, Romania, and as a structural fellow at the University Heart Center Graz, Austria (courtesy of a grant awarded by the Romanian Society of Cardiology).

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Acknowledgements

The authors wish to thank Dr Shaden Khalaf and Dr Cezar A. Iliescu from the Department of Cardiology, Division of Internal Medicine, M.D. Anderson Cancer Center, Houston, Texas, for their involvement in creating this article, especially for the clinical care of the patient at the highest standards and for helping us with PET–CT images and interpretations.

Consent: Informed consent for patient information and images to be published was provided by the patients in accordance with COPE guidelines.

Conflict of interest: PET images in this case use the HeartSee software for which the University of Texas receives licencing and royalty payments for its 510(k) applications K143664, K171303, K202679, and K231731. Unrelated to the current manuscript, N.P.J. and R.L.K. have patents pending on diagnostic methods for quantifying aortic stenosis and TAVI physiology, and on methods to correct pressure tracings from fluid-filled catheters. N.P.J. receives significant institutional research support from Neovasc/Shockwave (PET core lab for COSIRA-II, NCT05102019) and CoreAalst (PPG Global, NCT04789317). All other authors declare that they have no conflicts of interest.

Funding: None declared.

Data availability

The data underlying this article will be shared upon reasonable request to the corresponding author.

References

1. Achim A, Johnson NP, Liblik K, Burckhardt A, Krivoshei L, Leibundgut G. Coronary steal: how many thieves are out there? *Eur Heart J* 2023;**44**:2805–2814.
2. Gould KL, Kirkeeide R, Johnson NP. Coronary branch steal: experimental validation and clinical implications of interacting stenosis in branching coronary arteries. *Circ Cardiovasc Imaging* 2010;**3**:701–709.
3. Lipton MJ, Barry WH, Obrez I, Silverman JF, Wexler L. Isolated single coronary artery: diagnosis, angiographic classification, and clinical significance. *Radiology* 1979;**130**:39–47.
4. Johnson NP, Gould KL. Integrating noninvasive absolute flow, coronary flow reserve, and ischemic thresholds into a comprehensive map of physiological severity. *JACC Cardiovasc Imaging* 2012;**5**:430–440.