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CASE REPORT
Coronary intervention

A case report of non-dominant right coronary artery occlusion: not always benign!

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Background Non-dominant right coronary artery (RCA) occlusion is uncommon and usually affects a small area of the myocardium. Rarely, it can complicate fatal outcomes such as shock, cardiac arrest, bradyarrhythmia, or tachyarrhythmia.

Case summary A 50-year-old man with no significant medical history presented with ventricular fibrillation (VF) cardiac arrest. He required prolonged cardiopulmonary resuscitation and multiple defibrillation shocks to achieve return of spontaneous circulation. ST elevation was noted on inferior leads. Due to refractory VF, extracorporeal membrane oxygenation (ECMO) was initiated followed by coronary angiography which demonstrated 100% acute occlusion of proximal RCA (small non-dominant), 90% stenosis of ramus intermedius (RI), and 80% stenosis of obtuse marginal (OM) arteries. Left ventricular ejection fraction was 35%. Percutaneous coronary intervention (PCI) of the RCA was performed with drug eluting stent. He had excellent clinical recovery without any neurological deficits. The ECMO was weaned off and decannulated within three days. Guideline directed medical therapy was administered. He remained hemodynamically stable and underwent staged PCI of RI and OM to achieve complete revascularization.

Discussion Non-dominant RCA lesions are usually considered benign. However, when acute RCA occlusion results in cardiac arrest as seen in our patient, prompt revascularization is necessary. Treatment of cardiogenic shock with appropriate pharmacological and mechanical therapies is important, such as ECMO in our patient.

Keywords Non-dominant RCA • Cardiac arrest • Ventricular fibrillation • CAD • Acute coronary syndrome • Case report

ESC Curriculum 3.2 Acute coronary syndrome • 3.1 Coronary artery disease • 5.6 Ventricular arrhythmia • 6.4 Acute heart failure • 7.2 Post-cardiac arrest

Learning points

- Acute occlusion of non-dominant right coronary artery can result in fatal consequences.
- Immediate resuscitation and coronary revascularization are cornerstones of effective treatment.

Introduction

Coronary dominance is defined by the coronary artery giving rise to the posterior descending artery (PDA). In 80% of population, the right coronary artery (RCA) gives off the PDA, while 10% have left dominance (PDA arising from the left circumflex artery), and 10% are codominant.[1] Non-dominant RCA occlusion is uncommon and usually affects a small portion of myocardium. However, it can produce catastrophic outcomes in some individuals, including hypotension, shock, cardiac arrest, bradyarrhythmia, or tachyarrhythmia.[2] We discuss a case of non-dominant RCA occlusion that led to cardiac arrest.

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Summary figure

Presentation	Presented with VF cardiac arrest, ST elevation in inferior leads post ROSC
15 minutes	Recurrent VF requiring prolonged CPR and multiple defibrillation shocks with no ROSC
45 min	Cannulated and initiated on ECMO for refractory VF leading to ROSC
90 minutes	Percutaneous intervention of RCA with drug-eluting stent placement. LVEF 35%
Day 2-4	TTE showed LVEF of 65%, Neurologically intact, ECMO gradually weaned off
Day 5	Guideline-directed medical therapy introduced
Day 6	Antibiotics for hospital-acquired pneumonia
Day 7	Intact neurological function. He was discharged home with plans for outpatient cardiac rehabilitation.
Week 4	Staged PCI of ramus intermedius and obtuse marginal arteries

CPR=cardiopulmonary resuscitation; ECMO=extracorporeal membrane oxygenation; LVEF=left ventricular ejection fraction; PCI=percutaneous coronary intervention; RCA=right coronary artery; ROSC=return of spontaneous circulation; TTE=transthoracic echocardiogram; VF= ventricular fibrillation.

Case presentation

A 50-year-old non-smoker man with no significant past medical history presented with a witnessed out-of-hospital cardiac arrest. He had no prior known risk factors for coronary artery disease (CAD) and was not taking any medications. Immediate bystander cardiopulmonary resuscitation (CPR) was initiated by family members. Emergency medical services personnel arrived and found him unresponsive and pulseless. The initial rhythm was noted to be ventricular fibrillation (VF). He had a return of spontaneous circulation (ROSC) after CPR for two minutes, followed by one round of defibrillation (downtime 10 min). He was intubated on the scene and brought to the emergency room. Electrocardiogram post-ROSC showed normal sinus rhythm with ST elevation in inferior leads (*Figure 1*). After arrival at the emergency room, he had recurrent VF arrest prompting repeat CPR. He received five defibrillation shocks (up to 360 J) and amiodarone boluses (150 mg followed by 300 mg) with no ROSC. With ongoing CPR, due to refractory VF, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) was cannulated within the next 30 min. Following VA-ECMO initiation, ROSC was achieved (total downtime of 30 min). Amiodarone infusion was continued after ROSC. Bedside transthoracic echocardiogram (TTE) showed depressed left ventricular ejection fraction (LVEF) at 35%. Coronary angiography was performed, and it demonstrated 100% acute occlusion of the proximal RCA (non-dominant, small caliber vessel) (*Figure 2*), 90% stenosis of proximal ramus intermedius (RI), and 80% stenosis of obtuse marginal (OM3)

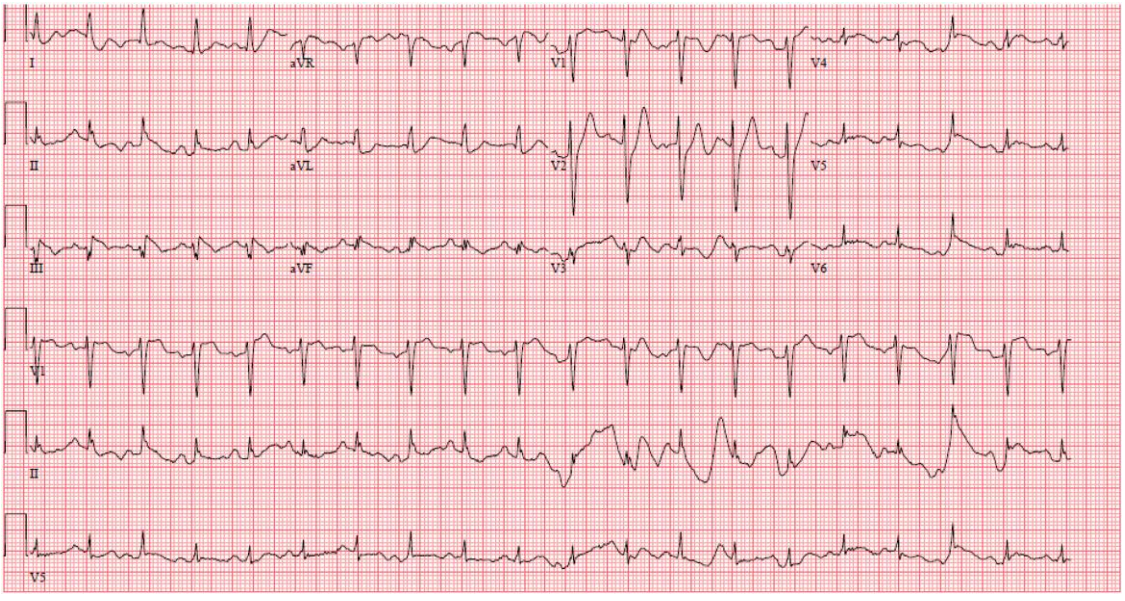

Figure 1 Initial ECG demonstrating ST elevation in inferior leads.

arteries (*Figure 3*). Given refractory VF and evidence of inferior STEMI on presenting ECG, percutaneous coronary intervention (PCI) of proximal RCA was performed with a 2.25 × 18 mm Resolute Onyx drug-eluting stent leading to Thrombolysis in Myocardial Infarction III blood flow (*Figure 4*). The patient was subsequently admitted to the cardiac intensive care unit. Initial labs were notable for white blood cells 22 200/μL (ref: 4000–11 000/μL), Creatinine 1.41 mg/dL (ref: 0.64–1.27 mg/dL), Troponin-I 0.04 ng/mL (ref: <0.04 ng/mL), lactic acid11.5 mmol/L (ref: 0.5–2.0 mmol/L), and anion gap of 19 mmol/L (ref: 4–15 mmol/L). Computed tomography of the head did not show evidence of anoxic brain injury. TTE after 24 h exhibited normal LVEF at 65%. The right ventricle was mild to moderately dilated with preserved systolic function and normal right atrial pressure (3 mmHg). Pulmonary artery systolic pressure could not be assessed due to insufficient tricuspid regurgitation on TTE. Dual antiplatelet therapy was administered with aspirin and clopidogrel. The mechanical ventilation and VA-ECMO were weaned off and decannulated within 2–3 days. After recovery from the acute phase and gaining consciousness, the patient reported that he had been having jaw pain for a few days before presentation; however, he denied chest pain or dyspnea. He did not have recurrent ventricular arrhythmia on telemetry after PCI. The hospital course was complicated by hypertensive urgency, Hemophilus influenzae pneumonia, and acute renal injury, all treated appropriately. Guideline-directed medical therapy was administered for CAD. The final medical therapy included aspirin 81 mg daily, clopidogrel 75 mg daily, atorvastatin 80 mg daily, carvedilol 25 mg twice daily, amlodipine 10 mg daily, and amiodarone 200 mg daily. The patient showed good clinical recovery within a few days and had no neurological deficit. He was discharged home with cardiac rehabilitation planned as an outpatient. He remained haemodynamically stable and underwent staged PCI of RI and OM3 within four weeks. He continued to have preserved LVEF (65%) on TTE and remained asymptomatic at 4-week and 6-month follow-ups.

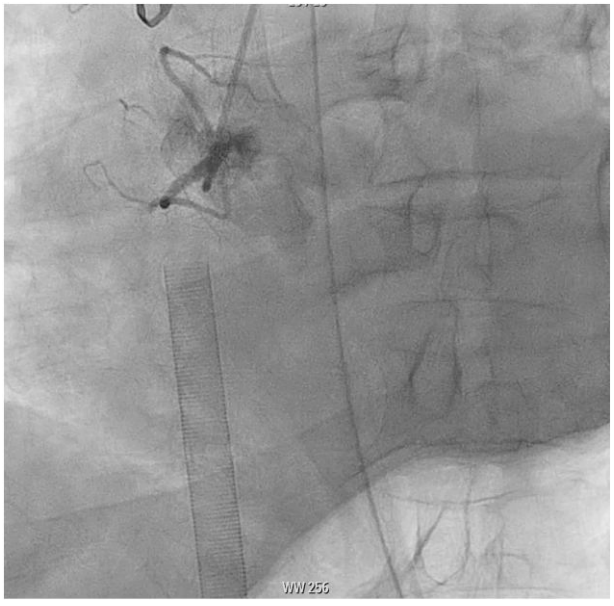

Figure 2 Coronary angiogram showing 100% acute occlusion of proximal RCA.

Discussion

Acute occlusion of non-dominant RCA usually affects a small territory of myocardium. Therefore, the clinical impact is generally minor. However, it may lead to fatal outcomes such as cardiogenic shock, cardiac arrest, bradyarrhythmia, or tachyarrhythmia in rare circumstances.

The basic pathophysiology behind these variable manifestations is ischaemia, albeit beginning in a small territory. Cardiogenic shock could result from right ventricular (RV) infarction; ventricular arrhythmia is plausible with ischaemia-induced ectopic focus; bradyarrhythmia could be associated with sinoatrial or atrioventricular node ischaemia.[3] In our

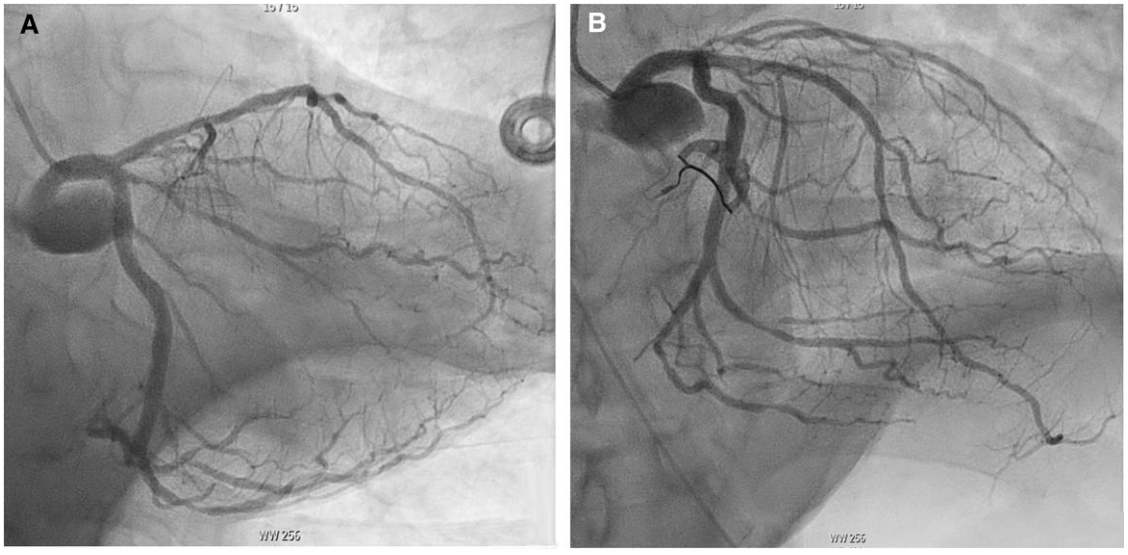

Figure 3 (*A*) Coronary angiogram in right anterior oblique cranial view showing 80% stenosis of ramus intermedius. (*B*) Coronary angiogram in right anterior oblique cranial view showing 90% stenosis of obtuse marginal 3 artery.

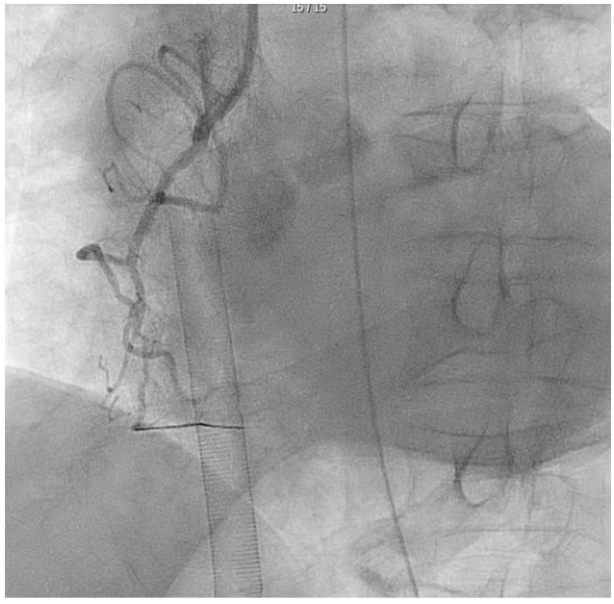

Figure 4 Coronary angiogram after the percutaneous intervention of RCA with 2.25 × 18 mm Resolute Onyx drug-eluting stent leading to TIMI-III blood flow.

patient, RV infarction was likely the culprit for circulatory collapse given acute total occlusion of RCA, even though it was a non-dominant vessel. Occlusion of RCA proximal to the origin of the RV branch likely explains the haemodynamic collapse. A handful of case reports document catastrophic outcomes following a non-dominant RCA occlusion.[2,4–6]

Devitt *et al.* reported a case where the patient suffered from a VF arrest due to non-dominant RCA occlusion. However, that patient had in-hospital cardiac arrest, unlike our case.[2] Some case reports highlight hypotension following RV infarction due to non-dominant RCA occlusion.[2,4] The clinical outcomes in such patients vary from rapid recovery to prolonged hospitalization and death.[5] Our patient presented with out-of-hospital cardiac arrest due to VF. Given the refractory nature of VF and fulminant cardiogenic shock, the ECMO was set up as a part of CPR facilitating ROSC. Immediate coronary angiography showed multivessel disease with 100% occluded non-dominant RCA, and PCI was performed on ECMO support. While other differentials were considered as the culprit for VF, such as myocarditis, infiltrative cardiomyopathy, or channelopathies, ischaemia was the most likely explanation in our patient in the setting of acute ECG changes with ST elevations in inferior leads. In addition to ischaemic evaluation, select patients with ventricular arrhythmia (VF or ventricular tachycardia) should be evaluated with cardiac magnetic resonance imaging and genetic testing. These tests were deferred in our patient as he did not have recurrent ventricular arrhythmia on telemetry after PCI of RCA. Although he had obstructive CAD in RI and OM3, RCA was totally occluded and, in the acute setting, considered to be the culprit lesion. He underwent staged PCI of RI and OM3 for complete revascularization four weeks after index presentation.

When such patients develop concomitant cardiogenic shock, inotropic or mechanical circulatory support such as ECMO may be necessary while coronary revascularization of the culprit lesion is accomplished. Once the acute phase is treated, guideline-directed medical therapy is paramount for the long-term recovery of myocardium.

Fortunately, our patient did not suffer from irreversible brain damage or disability, primarily attributed to immediate interventions, as stated above.

Lead author biography

Muhammad Asim Shabbir, MD, is a cardiovascular disease fellow at the University of Nebraska Medical Center, NE. He completed his Internal Medicine and chief residency at Albany Medical College, NY. His research interests include coronary intervention and mechanical circulatory support.

Consent: Informed written consent was obtained from the patient on the form provided by the EHJ-case report journal in accordance with COPE guidelines.

Conflict of interest: M.A.S., MD and A.J., MD: None. P.V., MD: Speaking fee from Medtronic, Abiomed, Opsens, Shockwave; Advisory board—Sanofi and Abiomed.

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Data availability

No new data were generated or analysed in support of this research.

References

1. Loukas M, Sharma A, Blaak C, Sorenson E, Mian A. The clinical anatomy of the coronary arteries. *J Cardiovasc Transl Res* 2013;**6**:197–207.
2. Devitt PA, Gaine SP, Magdy J, Coughlan JJ, Szirt R. Nondominant right coronary artery occlusion: small vessel, dramatic sequelae. *JACC Case Rep* 2022;**4**:156–160.
3. Gregory SA, Desai AS, Fifer MA. Isolated right ventricular infarction resulting from occlusion of a nondominant right coronary artery. *Circulation* 2004;**110**:e500–e5e1.
4. Karim MA, Wong C, Armbruster R, Windle J, Deligonul U. Importance of a non-dominant right coronary artery occlusion presenting as sudden cardiac death with prolonged right ventricular dysfunction and malignant arrhythmias. *Cathet Cardiovasc Diagn* 1995;**35**:257–261.
5. Franco JJ, Brown M, Bashir R, O'Murchu B. Acute anterior ST-elevation myocardial infarction and electrical storm secondary to nondominant right coronary artery occlusion. *Tex Heart Inst J* 2014;**41**:335–337.
6. Rao J, Mascarenhas DAN, Schiavone J. Nondominant right coronary artery occlusion and ventricular tachyarrythmias. *Catheter Cardiovasc Interv* 2002;**56**:53–57.