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CASE REPORT

INTERMEDIATE

CLINICAL CASE SERIES

Nondominant Right Coronary Artery Occlusion

Small Vessel, Dramatic Sequelae

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ABSTRACT

Nondominant right coronary artery (NDRCA) occlusion is rare and generally affects a small volume of myocardium. Despite this, NDRCA occlusion can result in dramatic clinical sequelae. These cases demonstrate the characteristic electrocardiographic findings and consequences of NDRCA occlusion, highlighting the importance of recognition of this pathologic condition to institute appropriate management. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:156–160) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Coronary arterial dominance is determined by the vessel that gives rise to the posterior descending artery, which supplies the posterior third of the interventricular septum. The right coronary artery is dominant in approximately 80% of the population. In 10% of the population, the posterior descending artery arises from the left circumflex artery,[1] rendering the right coronary artery (RCA) nondominant, with the remaining 10% of the population being codominant. Nondominant right coronary artery (NDRCA) occlusion is generally considered to be rare, although the exact incidence has not been accurately defined. Acute NDRCA occlusion has been associated with several clinical features, which include specific electrocardiogram (ECG) patterns, arrhythmia, and hypotension. We present 2 cases of NDRCA occlusion that demonstrate some of these clinical features and highlight the importance of recognition of this pathologic condition.

LEARNING OBJECTIVES

- To identify specific electrocardiographic patterns associated with nondominant right coronary artery occlusion.
- To recognize the dramatic clinical presentations that can be associated with nondominant right coronary artery occlusion, including arrhythmias and hypotension.

CASE DESCRIPTIONS

PATIENT 1. A 55-year-old woman presented to the emergency department with a 3-hour history of central chest tightness. She had no medical history and

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was a current smoker. Physical examination was unremarkable and her vital signs were stable. The ECG initially showed ST-segment elevation in leads III, V_1, and V_2 (Figure 1), which resolved on serial tracings. A bedside transthoracic echocardiogram (TTE) showed normal left ventricular ejection fraction and no regional wall motion abnormalities. Given that the ECG changes had resolved and the patient was now pain free, it was elected to treat her as having a non-ST-segment elevation acute coronary syndrome while awaiting determination of her troponin levels. She was administered dual antiplatelet therapy (DAPT) and enoxaparin.

The patient experienced new-onset fast AF (180 beats/min), which was managed with metoprolol. She subsequently experienced a ventricular fibrillation cardiac arrest and promptly underwent defibrillation (Figure 2). The patients' rhythm returned to AF on the cardiac monitor after defibrillation. An amiodarone infusion was started, and the patient was transferred to the coronary care unit.

The patient subsequently experienced junctional bradycardia with hypotension (systolic BP: 60 mm Hg), which was managed with intravenous fluids and dobutamine. The patient subsequently reverted to a normal sinus rhythm and her hemodynamic data stabilized. The initial troponin level was normal at <14 ng/L. Serial data collected showed a rise in troponin: 92 ng/L at 6 hours and 227 ng/L at 12 hours, peaking at 1,083 ng/L at 24 hours.

Coronary angiography demonstrated a proximally occluded NDRCA (Video 1). Percutaneous coronary intervention was performed to the proximal RCA with 2 overlapping drug-eluting stents (Video 2). Subsequent cardiac magnetic resonance showed normal left ventricular ejection fraction and mild right ventricular (RV) hypokinesis. The patient recovered well and was discharged without any further complications with DAPT, bisoprolol, ramipril, and statin therapy. At her 6-week follow-up clinic visit, the patient was well.

PATIENT 2. A 68-year-old man presented with 1 hour of central chest pressure associated with diaphoresis. His background included type 2 diabetes mellitus treated with metformin. On presentation, the patient was hypotensive, with a blood pressure of 70/40 mm Hg. An ECG demonstrated sinus bradycardia (54 beats/min), 1.5 mm of ST-segment elevation inferiorly and anteriorly, with ST-segment depression in the high lateral leads (Figure 3).

TTE revealed normal left ventricular (LV) size and systolic function and a dilated RV with preserved systolic function. He was administered fluids, DAPT

ABBREVIATIONS AND ACRONYMS

AF = atrial fibrillation
DAPT = dual antiplatelet therapy
ECG = electrocardiogram
LV = left ventricle
NDRCA = nondominant right coronary artery
RCA = right coronary artery
RV = right ventricle
RVOT = right ventricular outflow tract
TTE = transthoracic echocardiogram

FIGURE 1 Patient 1: Electrocardiogram Demonstrating ST-Segment Elevation in Leads III, V_1, and V_2

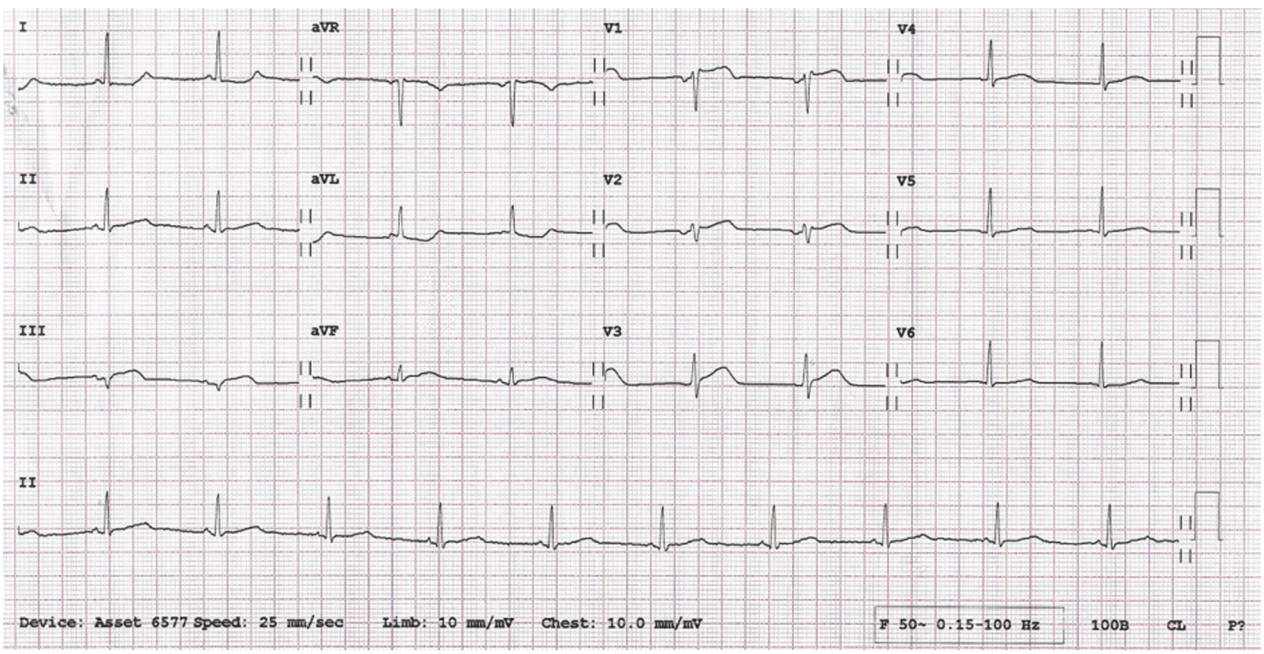
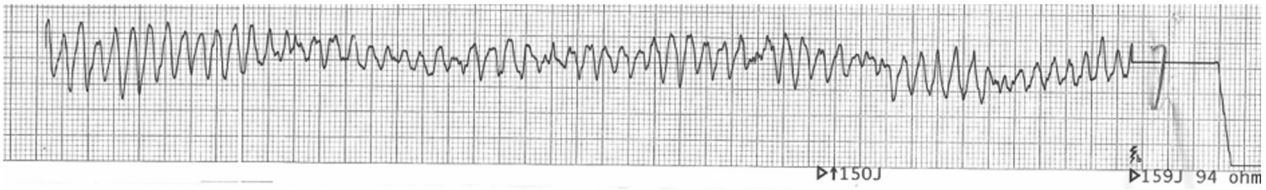


FIGURE 2 **Patient 1: Ventricular Fibrillation Which Was Promptly Defibrillated**



and intravenous heparin and underwent immediate cardiac catheterization.

Coronary angiography revealed a small NDRCA with a long segment of disease (99% stenosis, TIMI flow grade 1) in the proximal vessel, across the origin of a medium-caliber RV branch (bifurcation classification: Medina 1,1,1) (Video 3).

The RCA and RV branch were both wired, and the proximal RCA was stented into the larger marginal branch with the mid-RCA treated as the side branch. Kissing balloon inflation was performed, with a good final angiographic result (Video 4). After percutaneous coronary intervention, the patient's chest pain, hypotension, and ECG changes resolved. The patient was administered DAPT, atorvastatin, and ramipril. He was discharged 3 days later. Repeated TTE before discharge showed normal LV and RV size and function.

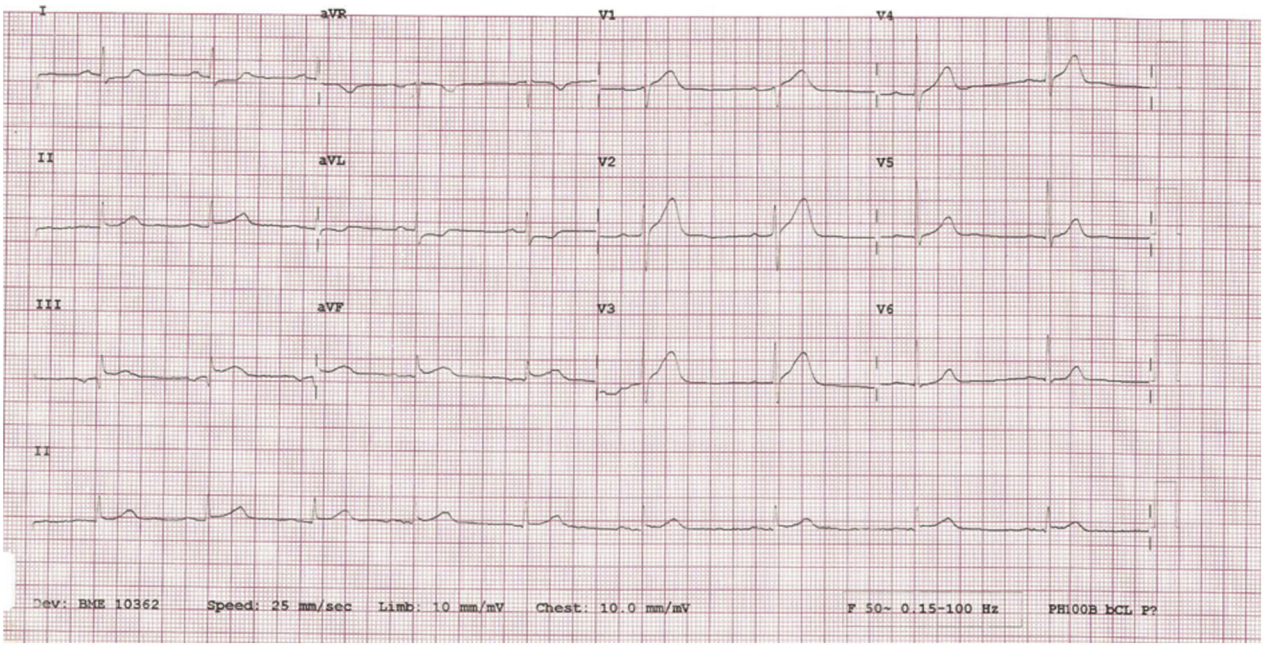
DISCUSSION

An analysis of previous case reports of NDRCA occlusion shows that 3 of the most common features identified appear to be subtle anterior ST-segment changes, arrhythmias, and hypotension. These features were present in the 2 cases described here, and we shall now discuss these features in more detail.

ANTERIOR ECG CHANGES. The ECG in the first case demonstrated ST-segment elevation in leads III, V_1, and V_2. In the second case there was ST-segment elevation inferiorly and anteriorly, with reciprocal depression in the high lateral leads. Several case reports have described ST-segment elevation in the anterior ECG leads in the setting of an NDRCA occlusion—changes that might make the clinician initially suspicious of an anterior infarct. Consideration of the anatomical areas supplied by an NDRCA helps explain these ECG findings. The posterior descending artery supplies the posterior third of the interventricular septum. Therefore, in a dominant right-sided system, the RCA supplies the inferior wall of the LV as well as the RV. When a dominant RCA is acutely occluded, ST-segment segment elevation in leads II, III, and aVF with reciprocal changes in leads I and aVL, is typically seen.[2] In most cases of proximal dominant RCA occlusion, injury of the anterior wall of the RV is not manifested on the precordial ECG leads because of the opposing forces of the accompanying posterior wall injury.[3] However, in an NDRCA infarct, the inferior wall is supplied by the left coronary circulation and will be spared, thus allowing the anterior RV wall injury to manifest on ECG, particularly with ST-segment elevation in leads V_1 and V_2, which are RV leads. If ST-segment elevation is seen anteriorly on the ECG and there are no corresponding regional wall motion abnormalities on echocardiogram anteriorly corresponding to the ECG changes, the clinician should consider an NDRCA occlusion. The 2 cases discussed here highlight the importance of considering NDRCA occlusion in patients with subtle anterior ECG changes.

ARRHYTHMIA. An NDRCA typically supplies important structures including the RV outflow tract (RVOT) (via the conus artery), the right atrium, the sinoatrial node (via nodal branches) and the RV (via the acute marginal branch). Interruption of the blood supply to any of these areas may cause either ventricular or atrial arrhythmia. Autonomic disturbances via damage to the sympathetic and vagal fibers at the RVOT may be a potential trigger for ventricular arrhythmias in the setting of an NDRCA occlusion. RV myocardial infarction has been shown to produce selective sympathetic and vagal denervation at viable sites in the RVOT and lateral peri-infarct regions, which may contribute to ventricular tachyarrhythmias.[4] Supraventricular arrhythmias may also result from sinus node dysfunction after an interruption of the blood supply to the SA nodal branch of the NDRCA or possibly via right atrial myocardial ischemia. The NDRCA often supplies both RA and RV myocardium. Ischemia of either RA or RV tissue may result in the generation of local electrolyte disturbances, reactive oxygen species, and re-entrant circuits, leading to arrhythmia. AF, ventricular tachycardia,

FIGURE 3 Patient 2: Electrocardiogram Demonstrating Sinus Bradycardia (54 beats/min) and 1.5 mm of ST-Segment Elevation Inferiorly and Anteriorly With ST-Segment Depression in the High Lateral Leads



and ventricular fibrillation have all been described in cases of NDRCA occlusion. The dramatic arrhythmogenic sequelae of NDRCA occlusion were highlighted in patient 1 in this report, who experienced AF, ventricular tachycardia, and junctional bradycardia in quick succession.

HYPOTENSION. NDRCA occlusion has also been associated with hypotension. If the RCA is occluded proximally, the resultant RV ischemia can lead to stiffness and dilation of the RV, with reduced RV diastolic filling. The resultant reduction in RV preload leads to a reduction in stroke volume and delivery of blood to the LV. Furthermore, as the RV dilates there can be interventricular septal shift into the LV, which can further reduce LV preload.[5] Hemodynamic instability is estimated to affect ≤50% of patients with RV infarction and may be a feature of NDRCA occlusion through acute RV marginal infarction.[6] Early recognition of RV infarction is important to guide management, with the subsequent avoidance of nitrates and the administration of inotropic agents and intravenous fluids. Another mechanism by which NDRCA occlusion may cause significant hypotension is through sinus node dysfunction and bradyarrhythmia, sometimes requiring temporary pacing.

CONCLUSIONS

NDRCA occlusion can be associated with several distinctive clinical features, including subtle anterior ST-segment elevation on electrocardiogram, hypotension, and both bradyarrhythmia and tachyarrhythmia. Recognition of these distinctive features may be helpful to allow physicians to promptly identify NDRCA occlusion and initiate appropriate management.

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KEY WORDS acute coronary syndrome, arrhythmia, hypotension, nondominant right coronary artery, ST-segment elevation myocardial infarction, sudden cardiac arrest

APPENDIX For supplemental videos, please see the online version of this article.