

Case Report

A Case of Electrical Right-Ventricular Infarction in the Absence of Clinical Right-Ventricular Failure

Robert T. Kay, MB, BCh, BAO, FRCPC,^{a,b} Blair J. O'Neill, MD, FACC, FRCPC,^{a,b}
Dylan Taylor, MD, FRCPC, FACC, FCCS, CCPE,^{a,b} and
Janek Manoj Senaratne, MD, MSc, FACC, FRCPC^{a,b,c}

^aDivision of Cardiology, Department of Medicine, University of Alberta, Edmonton, Alberta, Canada

^bDivision of Cardiology, Mazankowski Alberta Heart Institute, Edmonton, Alberta, Canada

^cDepartment of Critical Care Medicine, University of Alberta, Edmonton, Alberta, Canada

ABSTRACT

In the setting of acute coronary syndrome, right-ventricular (RV) infarction, which has significant clinical implications, can occur in conjunction with inferior left-ventricular (LV) infarction. In rare cases, RV infarction is isolated. We describe a case of isolated RV infarction identified based on previously described electrocardiogram findings in the absence of hemodynamic or imaging evidence of RV dysfunction. This case highlights the fact that RV transmural ischemia can exist in the absence of the clinical syndrome associated with RV infarction, which we hypothesize is related to the proportion of RV myocardium involved in the infarct, or conversely, the amount of myocardium protected through various mechanisms.

RÉSUMÉ

Dans le cadre du syndrome coronarien aigu, l'infarctus du ventricule droit, qui a des répercussions cliniques importantes, peut survenir conjointement avec un infarctus inférieur du ventricule gauche. Dans de rares cas, l'infarctus du ventricule droit est isolé. Nous décrivons un cas d'infarctus du ventricule droit isolé décelé à l'aide des résultats précédemment décrits d'un électrocardiogramme faite de résultats hémodynamiques ou d'imagerie indiquant une dysfonction ventriculaire droite. Ce cas souligne le fait qu'une ischémie transmurale du ventricule droit peut survenir même sans syndrome clinique associé à l'infarctus du ventricule droit, ce qui s'explique, selon notre hypothèse, par la proportion de myocarde ventriculaire droit touché par l'infarctus ou, à l'inverse, la quantité de myocarde protégé par divers mécanismes.

Myocardial infarction (MI) is defined as irreversible myocyte death secondary to lack of oxygen. The clinical definition of MI requires abnormal cardiac biomarkers, along with symptoms and evidence of acute myocardial ischemia. At presentation, the electrocardiogram (ECG) is the most readily available diagnostic tool for ischemia. ECGs record electrical activity of myocytes during the cardiac cycle. ECG patterns associated with ischemia include T-wave inversion, tall T-waves, ST-segment depression, and ST-segment elevation (STE).

ST-segment changes from myocardial ischemia occur due to adenosine triphosphate depletion and increased

extracellular potassium, resulting in membrane depolarization from baseline.¹ In transmural ischemia, the net depolarization at rest is away from the epicardium, causing lower-voltage T-Q segments and therefore by convention relative STE.¹ These changes occur quickly once adenosine triphosphate is depleted.¹ This knowledge of ECG changes allows STE to localize the ischemic cardiac territory. The most common anatomical designations on standard 12-lead ECGs are anterior, lateral, inferior, and posterior LV wall infarctions. Right-ventricular (RV) infarction accompanies up to 18% of inferior MIs, and identification of ECG findings often requires the addition of right-sided leads.²⁻⁴ The previously described clinical syndrome of RV infarction includes hypotension, venous distension, and clear lung fields.⁴⁻⁶ These patients are also sensitive to nitroglycerin, which causes hypotension due to hemodynamic reliance on RV preload. RV infarction can result in cardiogenic shock even in the absence of LV dysfunction; the morbidity and mortality associated with cardiogenic shock as a consequence of RV infarction highlight the need for timely diagnosis.⁴⁻⁶

As isolated RV infarction is rare, ECG findings are not well described in large-scale studies.^{4,6} Animal models and

Received for publication September 18, 2021. Accepted November 12, 2021.

Ethics Statement: The research reported in this article has adhered to the relevant institutional ethical guidelines.

Corresponding author: Robert Kay, Division of Cardiology, University of Alberta Hospital, 2C2, Walter Mackenzie Health Sciences Centre, 8440-112 St, Edmonton, Alberta T6G 2B7, Canada. Tel.: +1-613-898-0104; fax: +1-780-407-6507.

E-mail: rtkay@ualberta.ca

Twitter: [@DrRtka](https://twitter.com/DrRtka)

See page 343 for disclosure information.

Novel Teaching Points

- STE in the precordial leads (V1-3) can be a marker of RV ischemia in RCA occlusion.
- Addition of both V3R and V4R increases the specificity for determining presence of RV involvement.
- ECG evidence of RV transmural ischemia can occur in the absence of the clinical syndrome associated with RV infarction.

in-human iatrogenic models of isolated RV infarction have provided some clarity. Chou et al. studied a dog model of RV infarction and recorded STE followed by poor R-wave progression and Q-waves in right sided chest leads.⁶ Morgera et al.² retrospectively correlated ECG findings with human autopsy findings of RV infarction and found that STE > 1.0 mm in V4R, or STE > 0.5 mm in V4R in combination with

0.5 mm STE in V1, was associated with a 100% specificity for RV infarction. The presence of Q-waves in both the V3R and V4R leads was also associated with 100% specificity, whereas Q-waves in V4R alone were 88% specific.³ Finally, van der Bolt et al.³ studied a human model of isolated RV free wall infarction in 9 patients with RV branch occlusion during coronary intervention and found that STE in V1-V3 and V4R were seen in all patients acutely.

In this report, we describe a case of RV infarction identified based on symptoms, biomarkers, and specific ECG findings, including previously described anterior-lead STE during acute presentation in the absence of a clinical syndrome in keeping with RV dysfunction.

Case

A 59-year-old male current smoker with a 30 pack-year history, on no medications, presented with typical cardiac chest pain while at work that had initially started 5 hours earlier. His family history included a grandfather who suffered

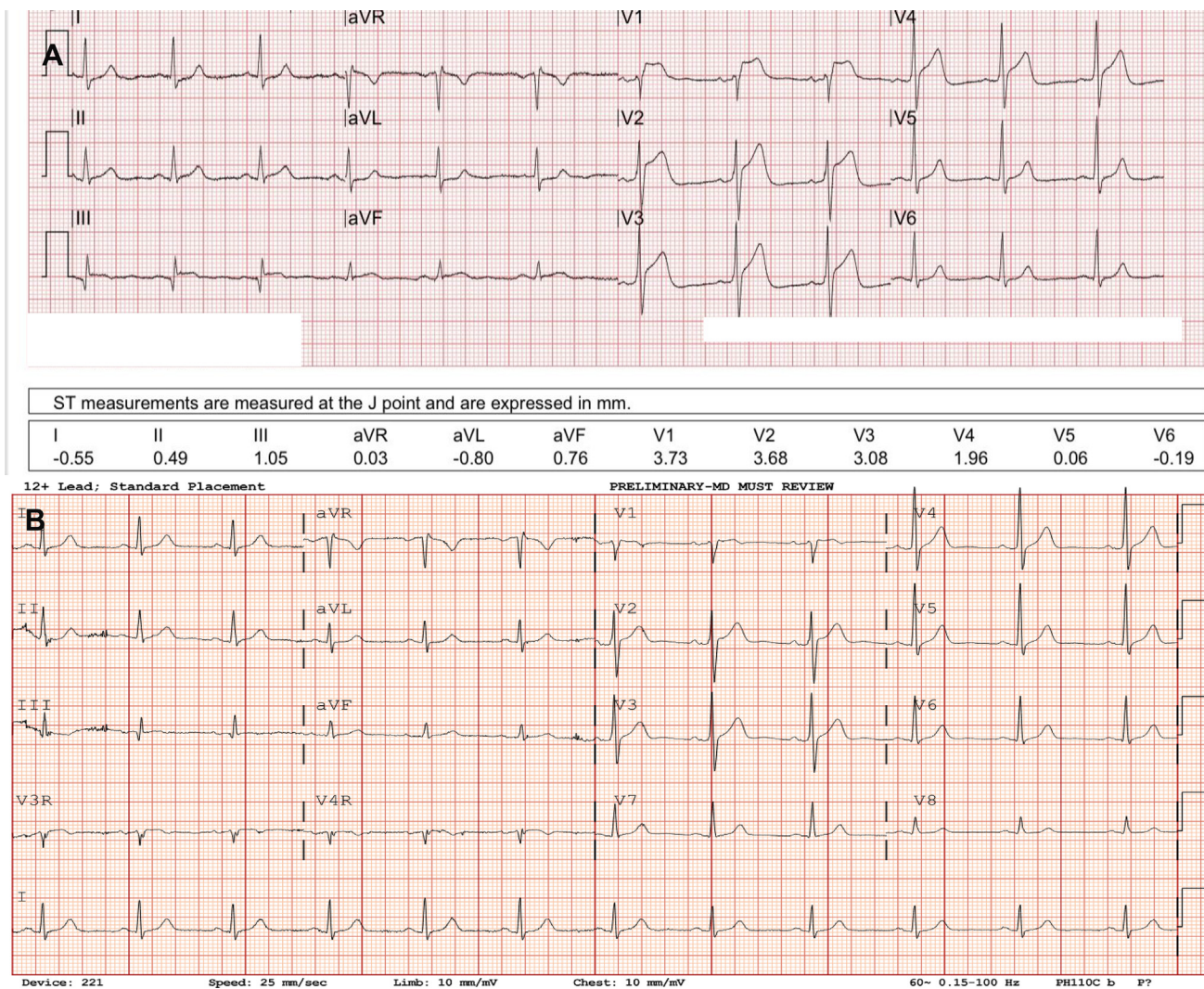





Figure 1. (A) Presenting electrocardiogram from emergency medical services with ST-segment elevation in III, V1-V3. **(B)** 16-lead electrocardiogram (post-percutaneous coronary intervention) Shown are q-waves in V4R, with T-wave inversion in V3R and V4R.

sudden cardiac death in his 30s. An ECG done with emergency medical services (EMS) showed a > 2 mm STE in V1-V3, along with a 1 mm STE in lead III, and 1 mm ST-segment depression in leads I and aVL (Fig. 1A). EMS transmitted the ECG to the on-call STE myocardial infarction (STEMI) service, which confirmed the diagnosis of STEMI. V4R was also reported to be elevated on a separate recording with EMS that was not transmitted. Given the patient's proximity to a primary percutaneous coronary intervention (PCI) center, the possibility of late presentation, and the presence of severe uncontrolled hypertension precluding fibrinolytic therapy, he was urgently transferred for primary PCI, with administration of aspirin (160 mg oral), ticagrelor (180 mg oral), enoxaparin (30 mg intravenous) and enoxaparin (85 mg [1 mg/kg] subcutaneous) en route. Due to concerns regarding RV infarction based on V4R, nitroglycerin was withheld.

At the primary PCI center, examination revealed an a-wave dominant jugular venous waveform 2 cm above the

sternal angle without Kussmaul's sign. Precordial auscultation revealed normal S1 and S2 without an S3, S4, murmur, or rub. There were clear lung fields and no peripheral edema. The patient underwent emergency coronary angiogram, which revealed a codominant coronary circulation with acute occlusion of the proximal right coronary artery (RCA) and no significant left coronary lesions (Fig. 2A/B; Supplemental Fig. S1; Videos 1-5 , view videos online). Left ventriculogram revealed normal LV contractility, without any regional wall-motion abnormalities (Video 6 , view video online). The RCA was revascularized with a drug-eluting stent (XIENCE Pro A, Abbott Cardiovascular, Plymouth, MN). The elapsed time from first medical contact to revascularization was approximately 130 minutes. Based on chest pain history, total ischemic time could have been as long as 7.5 hours, due to late presentation. Post-revascularization, RV branches appeared that were not previously evident (Fig. 2B; Video 7 , view video online). Of note, the patient was given nitroglycerin 200 mcg

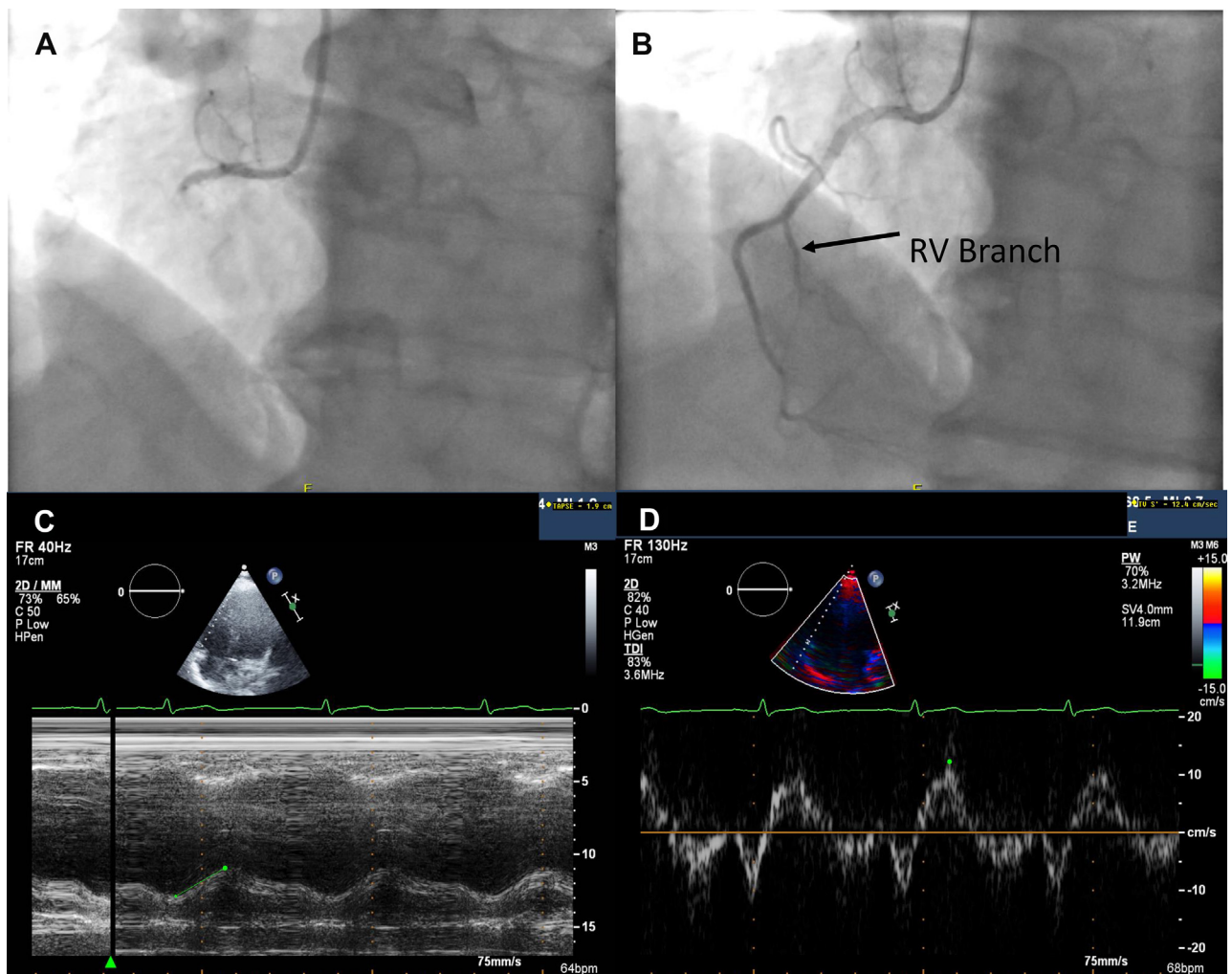



Figure 2. (A, B) Right coronary artery (RCA) angiogram pre- and post-percutaneous coronary intervention. (A) 100% proximal RCA occlusion. (B) RCA post-intervention, with the thrombolysis in myocardial infarction (TIMI) 3 flow revealing right-ventricular branches. (C, D) Transthoracic echocardiogram measurements of right-ventricular systolic function. (C) Tricuspid annular plane systolic excursion (TAPSE): 1.9 cm (normal: > 1.6 cm). (D) TVS': 12.6 cm/s (normal: > 9 cm/s).⁷

intravenously and 200 mcg intracoronary in the cardiac catheterization laboratory without hypotension.

He was transferred to the cardiac intensive care unit for ongoing management. A 16-lead ECG showed resolution of STE in leads III and V2-3, with persistence of 1 mm of STE in V1; right-sided leads revealed 1 mm of persistent STE with T-wave inversion in V3R and V4R and q waves in V4R. Posterior leads were unremarkable (Fig. 1B). Initial blood work revealed an elevated high sensitivity troponin I of 1520 ng/L. Transthoracic echocardiography revealed a nondilated right ventricle with preserved systolic function, based on visual and quantitative assessment. The inferior vena cava measurements were consistent with right atrial pressure of 3 mm Hg (Fig. 2C/D; Supplemental Fig. S2A/B; Videos 8-10  view videos online).

Overall, his hospital stay was uncomplicated, with institution of secondary prevention therapies including aspirin, ticagrelor, atorvastatin, bisoprolol and perindopril. Despite ECG evidence of RV infarction, he did not have any clinical or imaging signs of RV failure and no typical medication sensitivities. He was discharged home after 48 hours of monitoring in the cardiac intensive care unit.

Discussion

In this report, we describe a clinical case of STEMI with specific ECG findings showing RV infarction (STE in V1-3, V3R, V4R, and q-waves in V4R) on presentation, in the absence of a clinical syndrome in keeping with RV dysfunction. A review of the current literature indicates that there has not been a clear published clinical record highlighting separation of classical ECG findings of RV infarction from clinical sequelae. This case provides a clear clinical and imaging example of ECG findings associated with RV myocardial infarction (RVMI) in the absence of the clinical syndrome associated with RV dysfunction from acute RVMI. One limitation of our case is that we do not have a previous ECG showing absent q-waves in V4R, so we cannot know definitely that the q-waves are new. However, we feel that the other ECG findings, which have been described previously in the setting of controlled human and animal experiments of RV infarction, coupled with the proximal RCA occlusion on angiography limiting blood flow through the RV, clearly support the diagnosis of acute RV infarction.

Multiple mechanisms protect the RV from extensive infarction, including the following: increased oxygen supply due to systolic perfusion and lower oxygen demand due to smaller mass, and lower wall tension, resulting in a higher oxygen extraction reserve.^{4,5} Furthermore, collateral blood flow from the left coronary system to the right ventricle provides further protection,⁵ as outlined by Haupt et al., who studied postmortem hearts and found that the presence of collateral flow specifically through the moderator band artery from the left anterior descending perforators conferred protection from RVMI.⁸

In our case, we propose that these multiple mechanisms protected the right ventricle from extensive ischemia and infarction. We postulate that our distinct ECG findings were due to the proximal RCA occlusion restricting anterior RV

blood flow, whereas the clinical syndrome of RV dysfunction was avoided, as a large enough proportion of the RV myocardium was spared from ischemia. Therefore, clinical RV failure is related to total ischemic burden of the right ventricle and may not correlate with ECG findings.

In conclusion, this case provides real-world evidence that ECG changes in keeping with transmural RV ischemia can occur in the absence of the clinical syndrome associated with RV infarction. Therefore, management of STEMI with ECG evidence of RV involvement should be tailored based on the clinical picture at hand and not based solely on the presenting ECG.

Funding Sources

The authors have no funding sources to declare.

Disclosures

The authors have no conflicts of interest to disclose.

References

1. Okada JI, Fujii K, Yoneda K, et al. Ionic mechanisms of ST segment elevation in electrocardiogram during acute myocardial infarction. *J Physiol Sci* 2020;70:1-4.
2. Morgera T, Alberti E, Silvestri F, et al. Right precordial ST and QRS changes in the diagnosis of right ventricular infarction. *Am Heart J* 1984;108:13-8.
3. Van der Bolt CL, Vermeersch PH, Plokker HW. Isolated acute occlusion of a large right ventricular branch of the right coronary artery following coronary balloon angioplasty: the only true 'model' to study ECG changes in acute, isolated right ventricular infarction. *Eur Heart J* 1996;17:247-50.
4. Forman MB, Goodin J, Phelan B, et al. Electrocardiographic changes associated with isolated right ventricular infarction. *J Am Coll Cardiol* 1984;4:640-3.
5. Cohn JN, Guiha NH, Broder MI, et al. Right ventricular infarction. Clinical and hemodynamic features. *Am J Cardiol* 1974;33:209.
6. Chou TC, Fowler NO, Gabel MA, et al. Electrocardiographic and hemodynamic changes in experimental right ventricular infarction. *Circulation* 1983;67:1258-67.
7. Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography: endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr* 2010;23:685-713.
8. Haupt HM, Hutchins GM, Moore GW. Right ventricular infarction: role of moderator band artery in determining infarct size. *Circulation* 1983;67:1268-72.

Supplementary Material

To access the supplementary material accompanying this article, visit *CJC Open* at <https://www.cjcopen.ca/> and at <https://doi.org/10.1016/j.cjco.2021.11.005>.