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Anomalous Origins of Right Coronary Artery from the Left Coronary Sinus in Wolff-Parkinson-White Syndrome

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Ethical approval
This study was approved by Kangbuk Samsung Hospital's Institutional Review Board (IRB number: 2020-06-011).

Conflict of Interest
The authors have no financial conflicts of interest.

Anomalous coronary artery origin is a rare condition with approximately 1% incidence among the general population undergoing computed tomography (CT) angiography.[1)]

We report a rare case of a previous healthy 14-year-old boy with a coronary artery anomaly incidentally found during a fever work-up. He had no past history or family history of heart disease including sudden cardiac death. He never experienced symptoms such as chest pain, palpitation, dizziness, syncope, or dyspnea on exertion. On echocardiography, the right

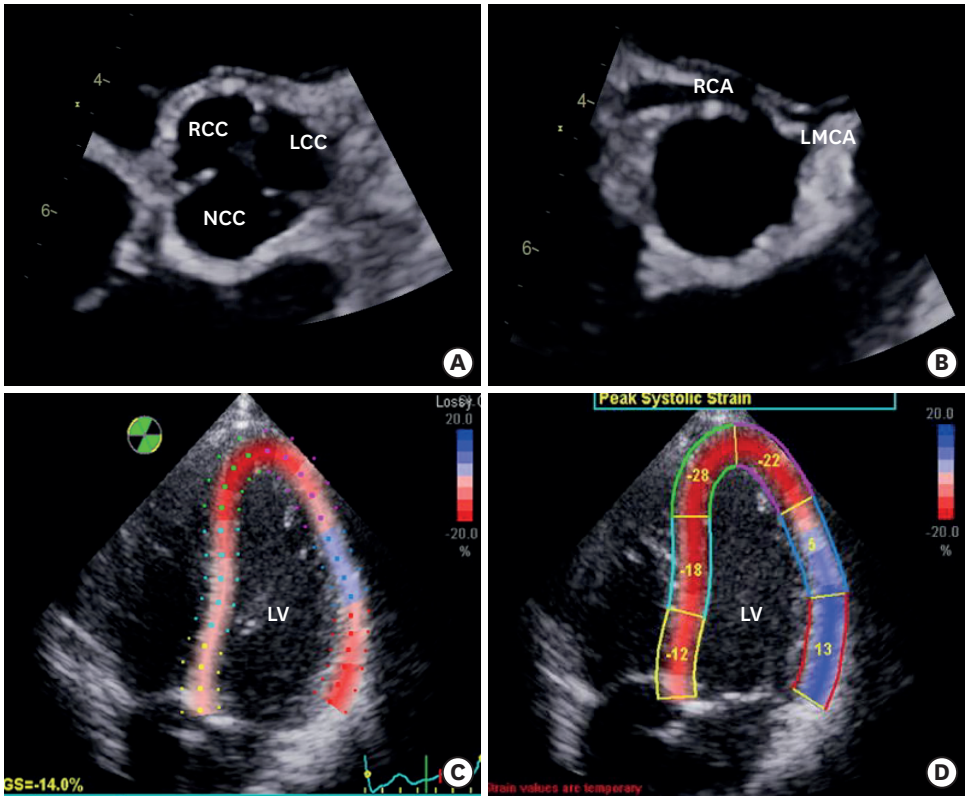

Figure 1. Echocardiography shows the aorta has 3 cusps (A; diastolic phase). The RCA seems to originate from the left coronary cusp (B; systolic phase). On strain, left lateral wall motion was decreased more than other areas. Global strain values = 14% (C, D).
RCC: right coronary cusp, LCC: left coronary cusp, NCC: non-coronary cusp, RCA: right coronary artery, LMCA: left main coronary artery, LV: left ventricle.

Author Contributions

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coronary artery (RCA) appeared to originate from the left coronary sinus and ran between the pulmonary artery and aorta, termed as "anomalous origins of the right coronary artery from the left coronary sinus (AORL)" (**Figure 1**). Enhanced coronary CT also showed the inter-arterial course of the AORL, which means the RCA ran between the pulmonary artery and aorta. The ostiums of the RCA and left main coronary artery (LMCA) were separated. Also, no significant (> 50%) luminal stenosis, slit-like ostium, or acute angle take-off were observed (**Figure 2**).

On 12-lead electrocardiography, compared to **Figure 3** shows a shorter PR interval and wider QRS accompanying delta waves which indicates Wolf-Parkinson-White (WPW) syndrome. To examine arrhythmia and ischemic effects, we performed 24-hour Holter monitoring, a modified Bruce protocol treadmill test, and stress echocardiography. Frequent premature ventricular contractions (373 beats) were observed on 24-hour Holter monitoring with 3 ventricular couplets, 8 bigeminy, and 2 trigeminy runs. There was no ventricular tachycardia. During the treadmill test, the patient did not complain of chest pain. But, down sloping ST depression at exercise phase 3 indicated myocardial ischemia. Wide complex rhythms with delta waves also appeared during the resting phase with ST changes (**Figure 4**). But no evidence of focal ischemia was noted on the stress echocardiography.

Though AORL is generally benign and asymptomatic, several cases with sudden cardiac death have been reported.[2)3)] Also, sudden cardiac death has been reported in patients with WPW

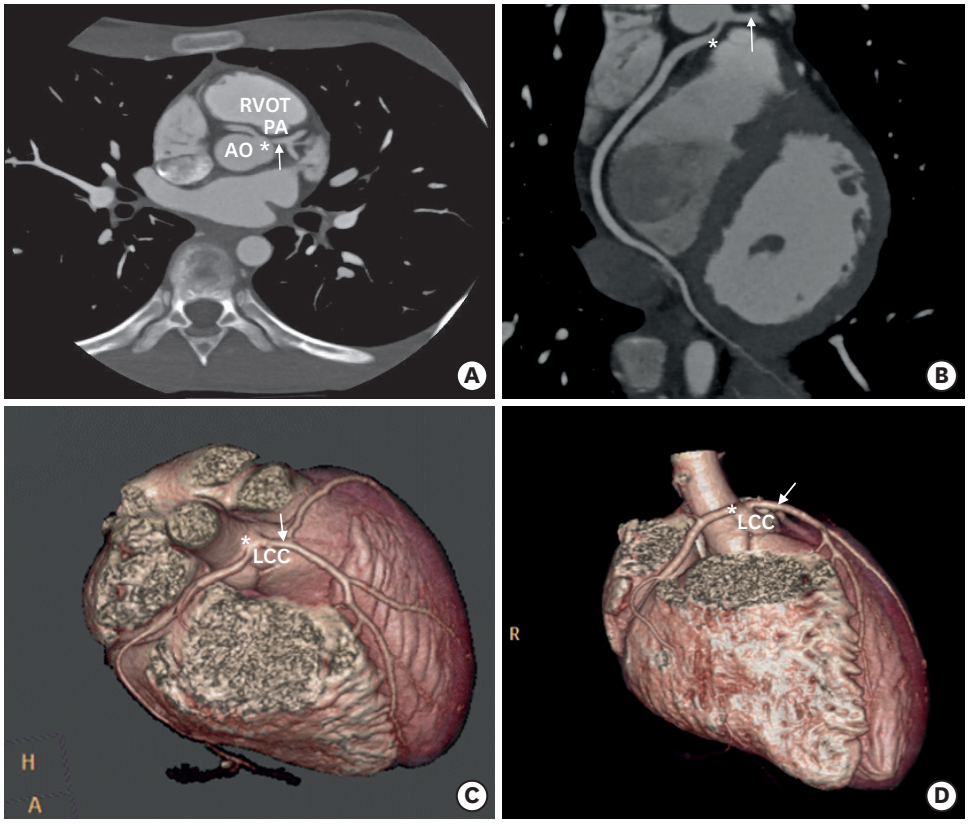

Figure 2. Coronary CT shows the RCA (asterisk) and LMCA (arrow) originate from the same coronary cusp, the LCC. The RCA runs between the pulmonary artery and aorta without significant luminal narrowing (A, B). The 3-dimentional reconstruction of a CT image showing the AORL (C, D).
CT: computed tomography, RCA: right coronary artery, LMCA: left main coronary artery, AORL: anomalous origins of the right coronary artery from the left coronary sinus, PA: pulmonary artery; RVOT: right ventricle outflow tract; AO: aorta, LCC: left coronary cusp.

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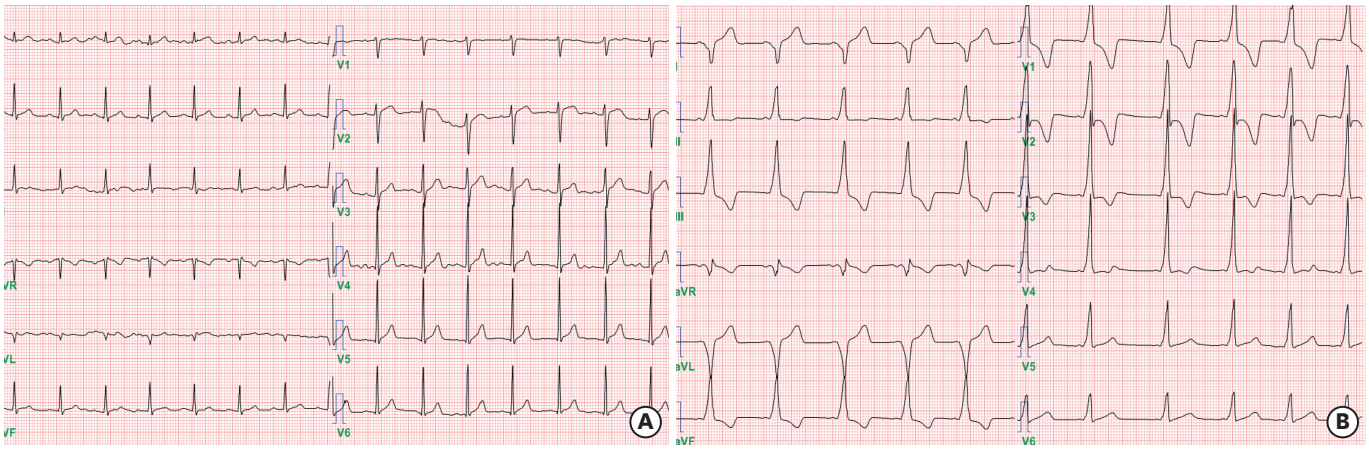

Figure 3. Electrocardiogram results. On HD #1. Normal sinus rhythm (A), HR 90 bpm, PR 0.176 seconds, QRS 0.090 seconds. During blood sampling on HD #3 (B), wide and regular QRS complexes are shown with delta waves. PR interval (0.112 seconds) is shorter than before and QRS (0.170 seconds) is widened. Nonspecific ST changes are seen on V2.
HD: hospital day, HR: heart rate.

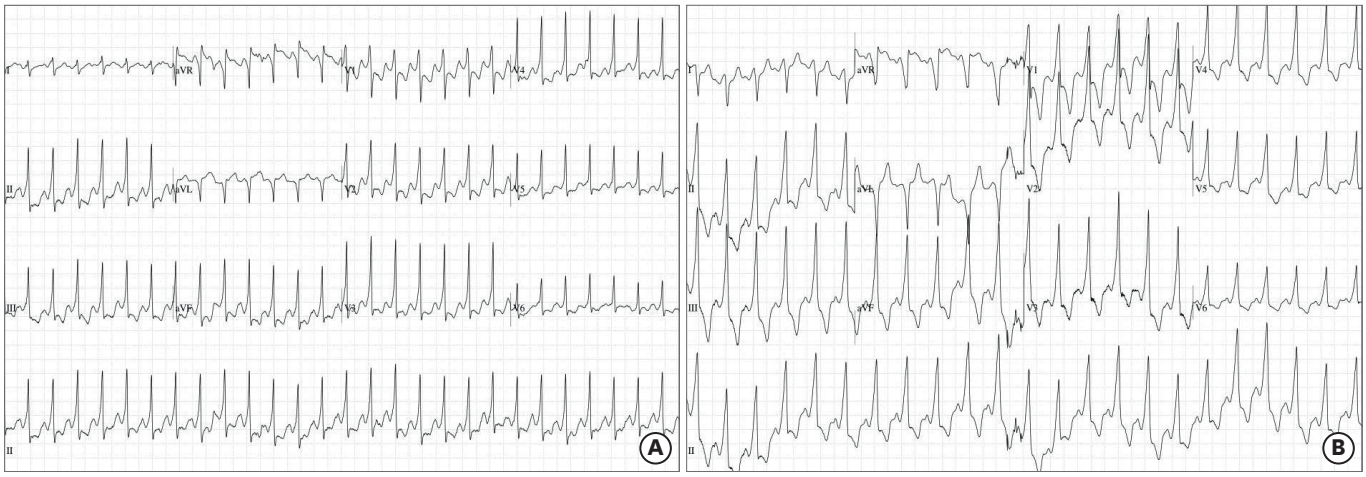

Figure 4. Treadmill test HD #6. ST depression on phase 3 (A). Wide QRS complexes with delta waves during resting phase with ST changes (B).
HD: hospital day.

syndrome.[4)5)] However, no statistical survey has been done to investigate these more aggressive effects on sudden cardiac death and no definite treatment guidelines that address these two combined abnormalities (AORL and WPW syndrome) have been proposed. So, it is highly challenging to make individualized treatment decisions. After discussion, we decided to observe the patient closely in the outpatient setting without intervention. We are planning to conduct further evaluation if the patient reports any newly appearing cardiac symptoms. To obtain more information, an electrophysiology study, intravascular ultrasound imaging, coronary angiography, and exercise scintigraphy would be helpful to determine appropriate management.

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