

# A Case of Anomalous Origin of the Right Coronary Artery from the Left Sinus of Valsalva

— Special Reference to PTCA Procedures and Aspirin Effect to Failed PTCA of the Anomalous Coronary Artery —

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**An unusual case of a 53-year-old male with a right coronary artery originating from the left sinus of Valsalva is presented. Despite acute myocardial infarction due to occlusion of the aberrant right coronary artery (RCA), PTCA was not immediately performed. After 81 mg/day of aspirin had been administered for 1 month, PTCA to the subtotal stenosis of the RCA resulted in failure due to poor deployment of the guiding catheters complicated by withdrawal and uncrossing of a guiding wire. However, complete recanalization occurred after giving the patient 162 mg/day of aspirin for 8 months. Low dose aspirin was effective in recanalizing the subtotal stenosis after failed PTCA.**

**Key words:** aberrant right coronary artery ; PTCA ; subtotal stenosis ; aspirin ; recanalization

## Introduction

Anomalous origin of the right coronary artery from the left sinus of Valsalva is an uncommon congenital condition and its incidence is 0.07-0.19% angiographically<sup>1,4)</sup> and 0.004% at autopsy<sup>5)</sup>. However, with the recent increased use of coronary angiography, more patients with this anomaly have been detected, some of whom have undergone percutaneous transluminal coro-

nary angioplasty (PTCA)<sup>6,7)</sup>. Currently, ticlopidine and/or aspirin is frequently used for cases that underwent PTCA or stenting for antiplatelet and antithrombin effects. Reportedly, higher daily doses (900-1500 mg) of aspirin are not more effective than lower doses (75-325 mg) for patients undergoing angioplasty, and aspirin alone, or in combination with dipyridamole, prevents early and late occlusion of the aortocoronary vein graft<sup>8)</sup>. We herein report a rare case of anomalous origin of the right coronary artery from the left sinus of Valsalva. Following an unsuccessful PTCA, late recanalization occurred after administering low dose aspirin alone. The PTCA procedure and approach are discussed, as well as the effect of aspirin.

## Case Report

A 58-year-old male with obesity, hypertension, hyperlipidemia and a long smoking history was admitted to our hospital with dyspnea and substernal chest pain, revealing 0.1 mV of ST depression in leads I, aVL and V<sub>4</sub> - V<sub>6</sub>, and a rS pattern in leads III and aVF on electrocardiography (ECG) (Fig. 1). No serum biochemical abnormality was evident. The patient received 1.5 mg/hr of isosorbide dinitrate (ISDN) and 500 IU/hr of heparin sodium intravenously without emergency coronary angiography.

The next day, the white blood cell count was 9100/ $\mu$ l, GOT 57 IU/l, LDH 342 IU/l, CPK 491 IU/l, and ECG showed QS in lead aVF (Fig. 1). Coronary angiography 19 hours after admission showed no significant stenosis of the left anterior descending coronary artery (LAD). After failing to detect RCA in the

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right sinus of the Valsalva with a Judkins-type right-6F-4cm (JR6F-4) catheter via the right femoral artery, it was demonstrated with a Judkins-type left-6F-4cm (JL6F-4) catheter and left ventriculography (LVG), revealing its orifice anteriorly above the LCA orifice in the left sinus of Valsalva (Fig. 2-A,B). This case was diagnosed as acute myocardial infarction due to occlusion of the proximal RCA, which originated anomalously from the left sinus of Valsalva. Intravenous infusions of ISDN and heparin sodium were continued for an additional 24 hours without intracoronary thrombolysis (ICT) or PTCA.

After orally administering 81 mg/day of aspirin and 40 mg/day of ISDN for 1 month, coronary angiography demonstrated thrombolysis in myocardial infarction (TIMI) grade 0 flow of the RCA with reduced inferior wall motion (Fig. 3-A). One week later, informed consent was obtained, and we proceeded with PTCA to the TIMI grade 1 flow of the mid-RCA (Fig. 3-B), via

the right femoral artery with a JL7F-4 guiding catheter (Vista britetip, Cordis, U.S.A.) and a 0.014" wire (Hi-Torque Floppy, ACS, U.S.A.). Despite several ballooning attempts with a 1.5-mm x 20-mm and a 4.0-mm x 20-mm balloon catheter (Trackstar, Cordis, U.S.A.), the lesion did not sufficiently dilate (Fig. 3-C). We changed the guiding catheter to an AL7F-1 and an AL7F-1.5 (Wiseguide, Scimed Life System Inc., U.S.A.) because of poor engagement. The inadvertently withdrawn guiding wire could not cross the lesion, resulting in failed PTCA (Fig. 4-A, B). The patient was placed on 162 mg/day of aspirin and 40 mg/day of ISDN indefinitely.

Eight months after the failed PTCA, coronary angiography revealed TIMI grade 3, and excellent RCA recanalization with normal inferior wall motion (Fig. 4-C). The patient has shown an uneventful course for 3 years.

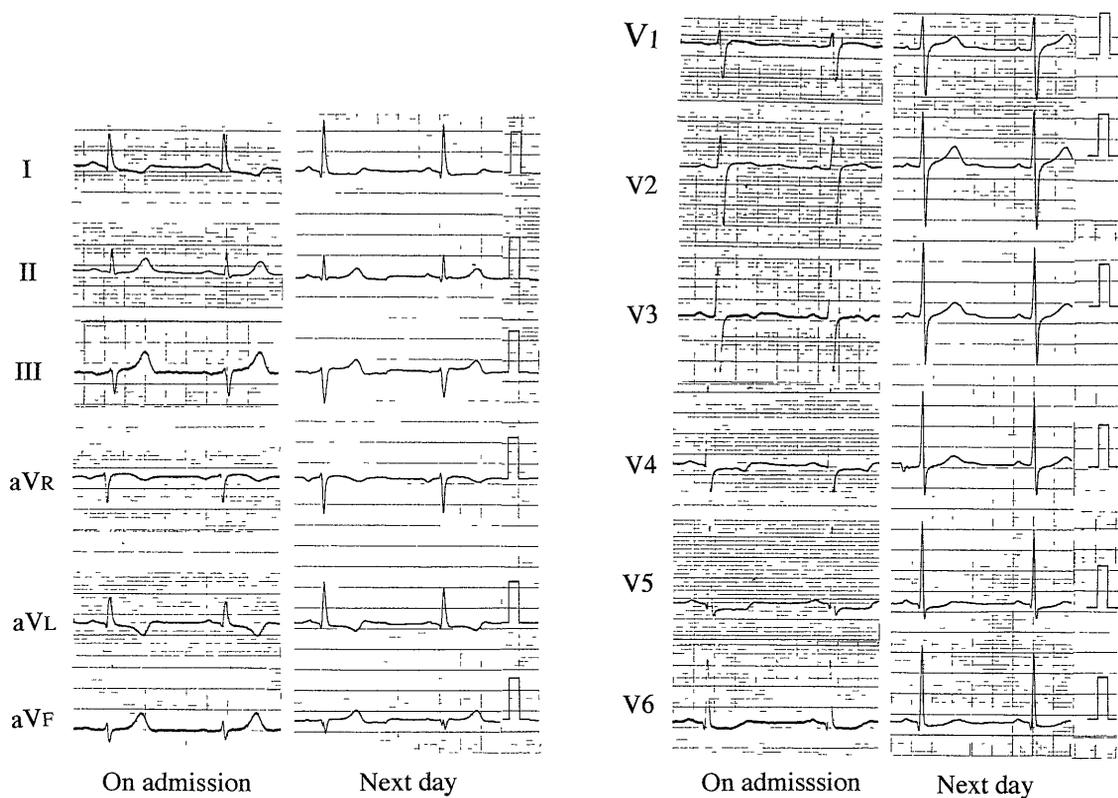
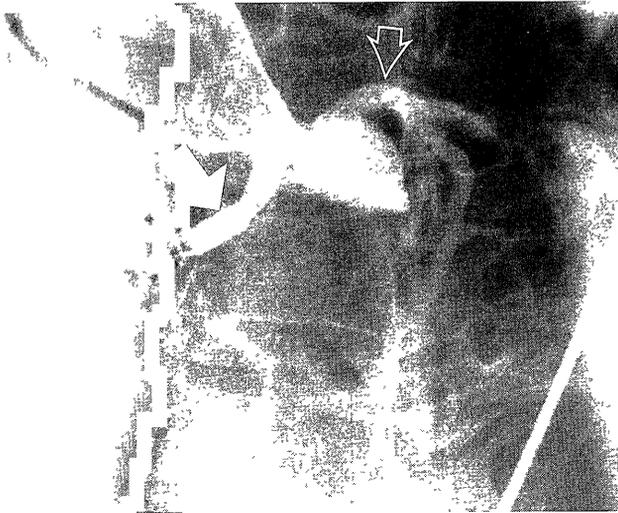


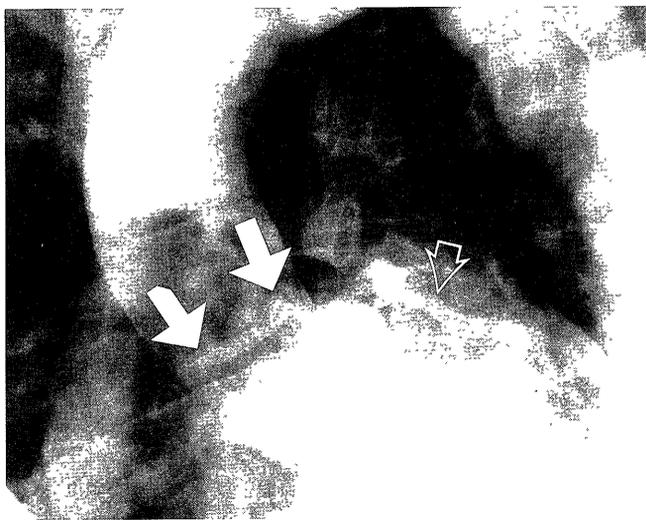
Fig. 1 Electrocardiography on admission and the next day.



A) The RCA originating from the left sinus of Valsalva shows complete occlusion and the LCA is faintly demonstrated.



A) The RCA shows complete occlusion associated with thrombus.



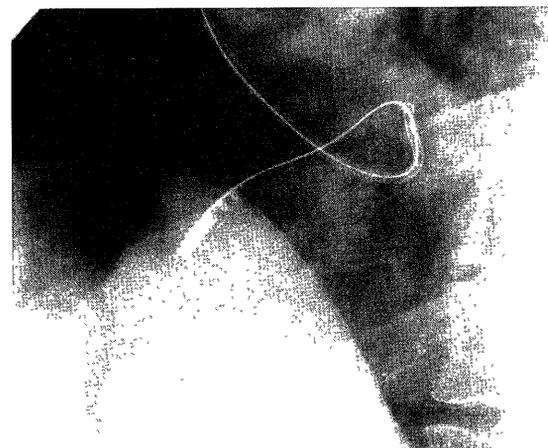
B) LVG shows high anterior takeoff RCA above the orifice of LCA.

The solid arrow demonstrates the RCA and the open arrow demonstrates the LCA.  
 LCA : left coronary artery  
 LVG : left ventriculography  
 RCA : right coronary artery

Fig. 2 Coronary angiography 1 day after admission.



B) One week later, TIMI grade 1 flow is evident.



C) PTCA is performed.

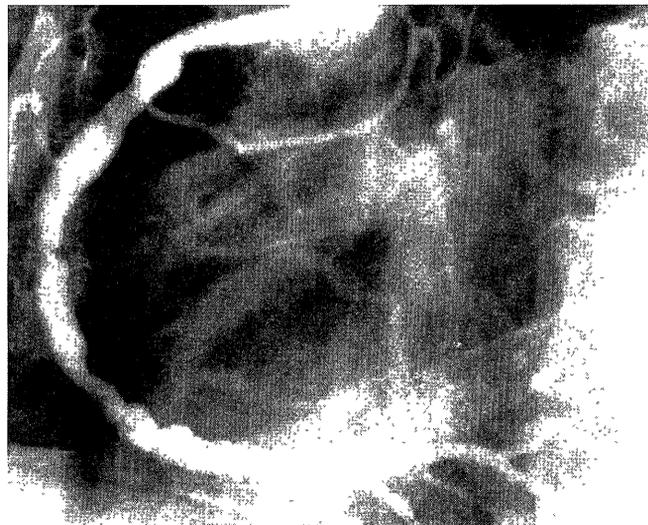
Fig. 3 Coronary angiography 1 month after AMI.



A) Guiding wire cannot cross the occluded lesion while changing guiding catheters.



B) PTCA results in failure without sufficient recanalization.



C) Complete recanalization of RCA occurs 8 months after failed PTCA.

**Fig. 4** Late recanalization after failed PTCA.

## Discussion

Although the origin of the RCA from the left sinus of Valsalva has been considered a minor congenital anomaly of no clinical significance<sup>9</sup>, current studies indicate that myocardial infarction, angina pectoris, syncope, ventricular tachycardia/fibrillation, and sudden death can be attributed to this aberrant anatomical condition<sup>9,11</sup>. The possible causes of the above dysfunctions include distal kinking<sup>5</sup> or compression of the ectopic artery between the aorta and the pulmonary trunk during exercise<sup>9,10</sup>, diminished flow into the RCA due to its upright slit-like ostium<sup>9</sup> and an acute angle origin<sup>10</sup>.

Although PTCA to this anomaly is technically difficult due to its abnormal anatomy<sup>12</sup>, it has been reported in patients with angina<sup>6</sup> and acute myocardial infarction<sup>7</sup>. Its success depends on the appropriate choice of balloon and a Judkins-type left or an Amplatz-type left guiding catheter in a stable position<sup>7,13</sup>. However, PTCA to the chronic subtotal stenosis with this anomaly is very rare. Rather than via the femoral approach, the right brachial artery approach should have been attempted in our case, because it has a good back-up due to improved angulation between the guiding catheter and the coronary ostium<sup>7</sup>.

The possible reasons why recanalization occurred

after failed PTCA are as follows : (1) the occluded lesion comprised thrombus alone without complete organization, calcification, tortuosity and significant stenosis ; (2) the duration of total occlusion was less than 35 days, and (3) oral administration of low dose aspirin in combination with ISDN may have been effective. We could not identify the exact date of recanalization because neither a 3 nor a 6-month follow-up angiography was performed. Since TIMI grade 0 improved to grade 1 flow within a week, recanalization possibly occurred within three months of the initial PTCA.

Although ticlopidine and aspirin have shown a synergistic effect in reducing platelet deposition<sup>14)</sup> and thrombin generation after PTCA<sup>15)</sup>, we gave low dose aspirin alone because its best risk-to-benefit and cost-to-benefit ratios were better than alternatives such as ticlopidine or warfarin, both of which reveal various side effects. Oral and intravenous administration of aspirin over 325 mg/day may reduce platelet aggregation and thrombosis formation more effectively than a lower dose<sup>16,17)</sup>. In contrast, our case showed that low dose aspirin (162 mg/day) recanalized the chronic subtotal stenosis naturally without any bleeding complications. This was supported by Winter et al. who reported 150 mg/day of oral aspirin or an intravenous dose of 100 mg/day can completely inhibit platelet aggregation and vasoconstriction, and a daily maintenance dose of 75mg is sufficient to effectively inhibit thromboxane A2 synthesis in long-term treatment<sup>18)</sup>.

In conclusion, this case showed that making an accurate diagnosis and therapeutic strategy for the aberrant coronary artery requires careful angiographic and left ventriculographic or aortographic observations with an appropriate approach and guiding catheter. Furthermore, low dose aspirin was effective to recanalize the chronic subtotal stenosis.

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