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Anomalous drainage of the great cardiac vein into the left atrial appendage: a two-case report

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ABSTRACT

Coronary venous system anomalies are rare structural variants that are usually detected incidentally. The great cardiac vein (GCV) normally drains into the coronary sinus (CS) through the valve of Vieussens, returning venous blood to the right atrium. Direct drainage of the GCV into the left atrial appendage (LAA) is an extremely rare anomaly, described in only a few reports. We present two female patients, aged 50 and 53 years, who underwent coronary computed tomography angiography (CCTA) for chest pain and in whom anomalous drainage of the GCV into the LAA was detected incidentally; in neither patient could a connection with the CS be demonstrated. Both patients were referred to cardiology; no intervention was indicated and clinical surveillance was recommended. At one year of follow-up neither had developed further cardiac symptoms, documented atrial arrhythmia or thromboembolic complications, and longer-term follow-up was not available. CCTA allowed the anomaly to be recognised and mapped three-dimensionally in both patients. Recognition of the anomaly is relevant to the planning of LAA-targeted interventions, transseptal approaches and coronary sinus-related procedures.

Keywords: Great cardiac vein, left atrial appendage, coronary venous anomaly, coronary computed tomography angiography, coronary sinus

INTRODUCTION

Anomalies of the cardiac venous system are rare congenital variations that usually follow an asymptomatic course and are detected incidentally during the investigation of other cardiac conditions.[1-3] Most of the coronary venous system drains into the right atrium through the coronary sinus (CS), which lies in the posterior left atrioventricular groove and opens into the right atrium adjacent to the orifice of the inferior vena cava.[1,4] The great cardiac vein (GCV) is the principal pathway of this drainage: it arises at the cardiac apex in the anterior interventricular sulcus, ascends parallel to the left anterior descending (LAD) artery, turns into the left atrioventricular groove alongside the left circumflex (LCx) artery, and continues as the CS at the level of the valve of Vieussens.[4,5]

The coronary venous system develops from the sinus venosus between the fifth and eighth weeks of gestation. As systemic venous return shifts progressively to the right, the left horn of the sinus venosus and the left common cardinal vein regress: the persisting proximal portion of the left sinus horn becomes the CS, whereas the obliterated left common cardinal vein persists as the oblique vein of the left atrium (vein of Marshall) and its fibrous remnant, the ligament of Marshall.[1,6,7] In parallel, the subepicardial venous plexus draining the developing myocardium establishes continuity with the CS. Failure of regression of the left cardinal system produces a persistent left superior vena cava (PLSVC); failure of separation between the CS and the left atrium produces an unroofed CS; and persistence of primitive connections between the subepicardial plexus and the left atrium-or failure of the GCV to establish continuity with the CS-may lead to coronary venous blood draining directly into the left atrium or the left atrial appendage (LAA).[1,2,8,9]

The spectrum of coronary venous anomalies also includes CS ostial atresia or stenosis, an absent or hypoplastic CS, and anomalous communications with the left-sided chambers.[1-3,5] Direct drainage of the GCV into the LAA is among the rarest of these variants and has, to our knowledge, been documented only in isolated case reports.[10-12]

We describe two patients in whom the GCV drained directly into the LAA with no demonstrable connection to the CS. We report the CCTA acquisition protocol and quantitative anatomical data, define the features that distinguish this anomaly from other vascular structures related to the LAA, and compare the two cases in order to illustrate the morphological variability of the entity.

CASE

Imaging Technique

All examinations were performed on a 64-detector-row CT scanner (Optima CT660, GE Healthcare, Milwaukee, WI, USA). Heart rate was controlled with oral metoprolol 50 mg once daily for five days before the examination and a further 50 mg 1-2 hours before scanning. Contrast-enhanced images were acquired with retrospective ECG gating after intravenous administration of 100 ml of iodinated contrast medium (Kopaq, 350 mg I/ml) through a right antecubital vein at a flow rate of 5 ml/s, followed by a saline chaser. Images were reconstructed at a section thickness of 0.625 mm over the 40-75% R-R interval. Post-processing included multiplanar and curved multiplanar reformation (cMPR), maximum intensity projection (MIP) and volume rendering (VR) on a dedicated GE AW workstation. Both examinations were reviewed in consensus by two radiologists with ten years' experience in cardiac imaging. Acquisition and reporting followed the recommendations of the Society of Cardiovascular Computed Tomography.[13,14]

Case 1

A 50-year-old female was referred for CCTA after cardiology assessment for atypical chest pain occurring both on exertion and at rest. She had no known coronary artery disease, hypertension or diabetes mellitus, and physical examination showed no cardiovascular abnormality.

Transthoracic echocardiography (TTE) showed a left ventricular ejection fraction (EF) of 60%, with no regional wall motion abnormality, valvular pathology or pericardial effusion. Cardiac biomarkers (high-sensitivity troponin I, CK-MB) and all other laboratory parameters were normal.

CCTA showed no obstructive coronary artery stenosis and was reported as CAD-RADS 1/E.[14] The LAD showed myocardial bridging in the anterior interventricular sulcus over a length of approximately 28 mm, with a superficial intramyocardial depth of 1-2 mm (**Figure 1**).

Evaluation of the venous system revealed a coronary venous drainage anomaly: the GCV ascended in the anterior interventricular sulcus alongside the LAD, continued into the left atrioventricular groove and terminated in the LAA instead of continuing as the CS (**Figure 2-4**). A thin-walled CS, approximately 2 mm in diameter, lay in the posterior atrioventricular groove and drained the mid- to distal-segment coronary veins (such as the middle cardiac vein and the posterior vein of the left ventricle) into the right atrium. No connection between the CS and the GCV was seen (**Figures 5** and **6**).

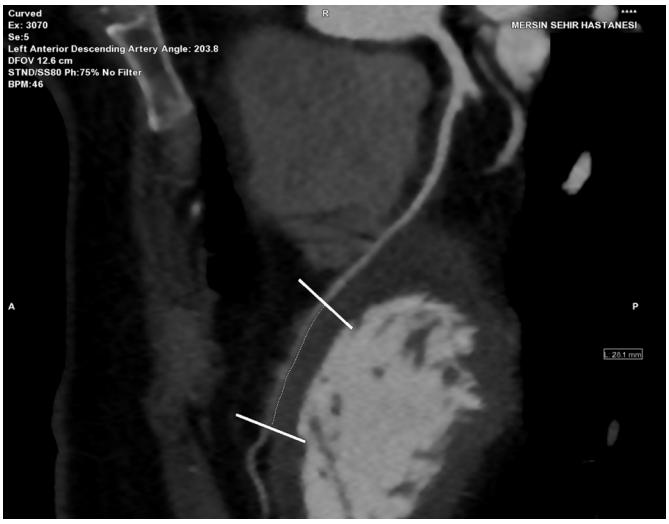

Figure 1. Case 1. cMPR CT image showing superficial myocardial bridging of the LAD coronary artery (28 mm in length, 1-2 mm intramyocardial depth)

DFOV: Display field of view, BPM: Beats per minute, cMPR: Curved multiplanar reformation, CT: Computed tomography, LAD: Left anterior descending

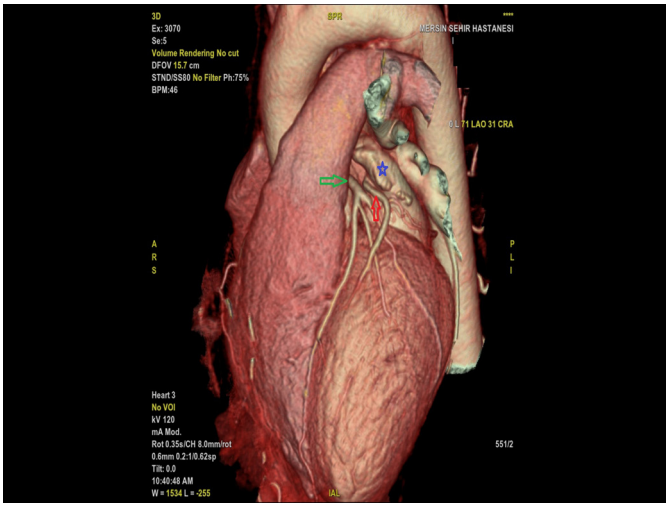

Figure 2. Case 1. Three-dimensional volume-rendered CT image. Blue star: LAA; red arrow: GCV; green arrow: left main coronary artery. The GCV terminates directly in the LAA

DFOV: Display field of view, BPM: Beats per minute, kV: Kilovolt, mA: Milliampere, Mod.: Modulation, Rot: Rotation, s: Second, mm: Millimeter, CH: Channel, sp: Spacing, AM: Ante meridiem, W: Window width, L: Window level, Vol: Volume, VOI: Volume of interest, LAO: Left anterior oblique, CRA: Cranial, LAA: left atrial appendage, GCV: Great cardiac vein

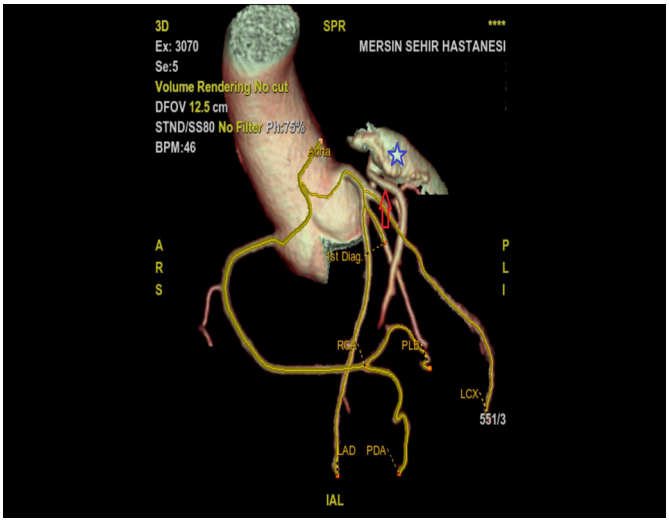

Figure 3. Case 1. Three-dimensional volume-rendered CT image in a complementary projection. Blue star: LAA; red arrow: GCV; green arrow: left main coronary artery

3D: Three-dimensional, Ex: Examination, Se: Series, SPR: Superior-posterior-right, DFOV: Display field of view, STND: Standard, Ph: Phase, BPM: Beats per minute, RCA: Right coronary artery, PLB: Posterolateral branch, LCX: Left circumflex artery, LAD: Left anterior descending artery, PDA: Posterior descending artery, CT: Computed tomography, LAA: left atrial appendage, GCV: Great cardiac vein

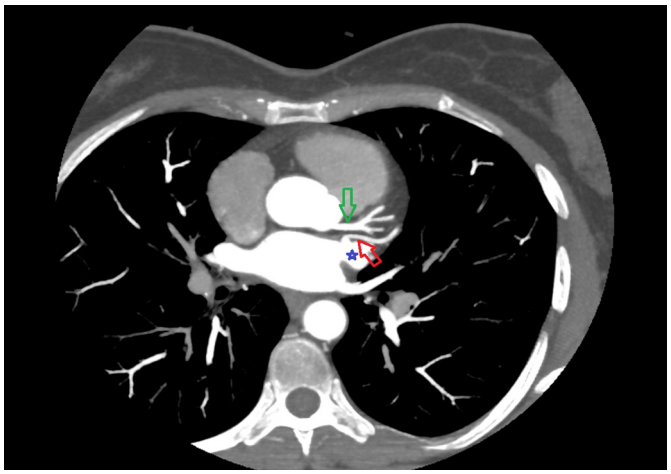

Figure 4. Case 1. Axial CT image. Blue star: LAA; red arrow: GCV; green arrow: left main coronary artery
CT: Computed tomography, LAA: left atrial appendage, GCV: Great cardiac vein

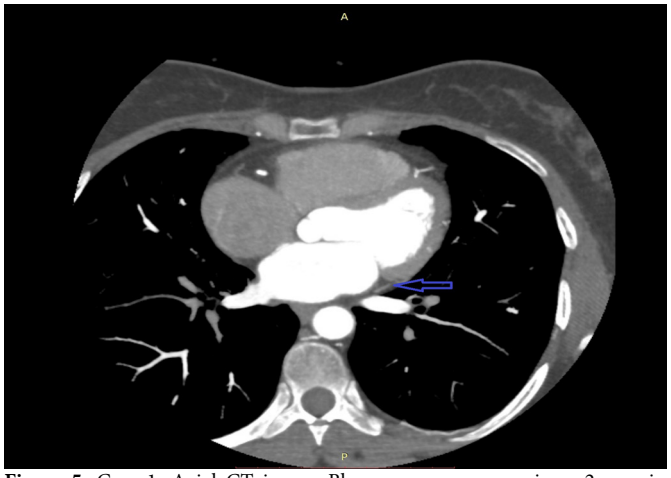

Figure 5. Case 1. Axial CT image. Blue arrow: coronary sinus, 2 mm in diameter, in the posterior atrioventricular groove
CT: Computed tomography

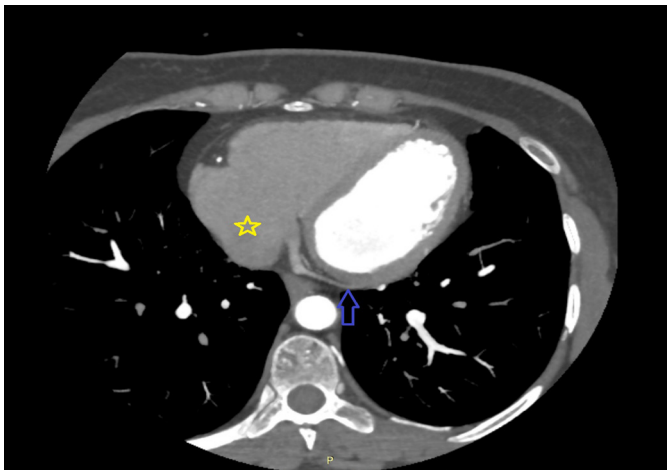

Figure 6. Case 1. Axial CT image, consecutive section following Figure 5. Blue arrow: coronary sinus; yellow star: right atrium
CT: Computed tomography

Quantitative measurements: the GCV measured 2.2 mm in the anterior interventricular sulcus and 2.3 mm immediately proximal to its termination in the LAA. The anomalous segment, measured from the point at which the vessel left the expected course of the CS to its termination, was 60 mm long. Because the study was acquired in the arterial phase, all venous measurements were taken along the segment in which luminal contrast filling of the vein could be followed, and the reported length corresponds to this opacified segment. The LAA was of chicken-wing morphology, with maximum dimensions (AP×ML) of 11×24 mm.[15]

Clinical course and follow-up: As the bridged LAD segment was superficial (1-2 mm), the consulting cardiologist did not consider it to account for the patient's atypical chest pain, and no further ischaemia testing (stress testing or invasive coronary angiography) was requested. The venous anomaly required no intervention and clinical surveillance was recommended. At one year the patient reported no recurrent chest pain or palpitations, and no atrial arrhythmia or thromboembolic event had been documented.

Case 2

A 53-year-old female was referred for CCTA after assessment for chest pain and exertional dyspnoea. She had no known cardiovascular disease and physical examination was unremarkable.

TTE showed an EF of 65%, with no valvular pathology, regional wall motion abnormality or intracardiac mass. All laboratory parameters, including cardiac biomarkers, were normal.

CCTA showed no obstructive coronary artery stenosis and was assessed as CAD-RADS 0.[14] A contrast jet consistent with a small secundum-type atrial septal defect (ASD) approximately 2 mm in diameter was identified at the mid-level of the interatrial septum (Figure 7).

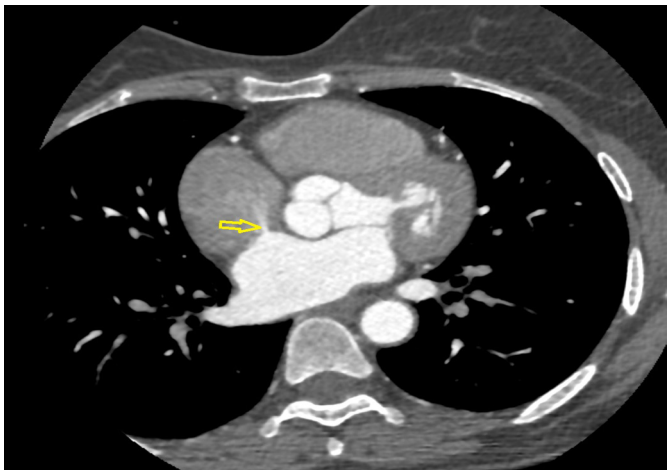

Figure 7. Case 2. Axial CT image. Yellow arrow: small secundum-type ASD
CT: Computed tomography, ASD: Atrial septal defect

Analysis of the venous system demonstrated an anomalous GCV with a tortuous course. After leaving the left atrioventricular groove the vessel passed through the space bounded anteriorly by the ascending aorta and posteriorly by the roof of the left atrium, and drained into the LAA (Figure 8-11). Because only an arterial-phase acquisition was obtained, luminal contrast filling was incomplete along the distal course of the vessel towards the interventricular septal region, so that the total length of the GCV could not be measured optimally and quantitative assessment was confined to the opacified segment. No hypoplasia, focal narrowing or interruption of the distal segment was identified; the appearance was attributed solely to suboptimal venous opacification in the arterial phase, and the

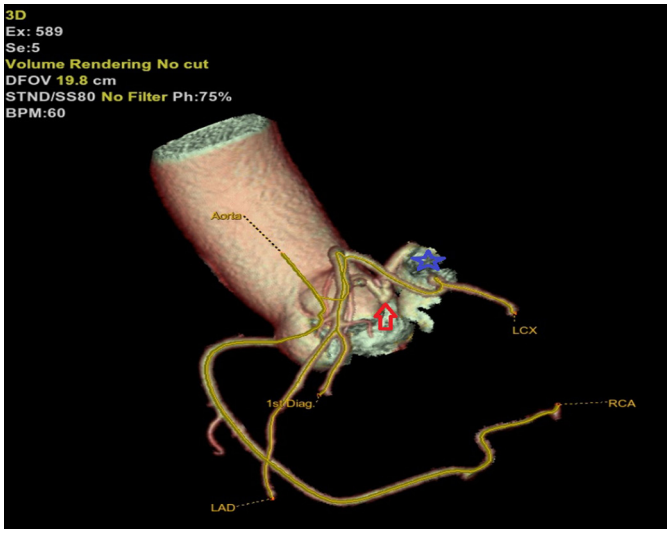

Figure 8. Case 2. Three-dimensional volume-rendered CT image. Blue star: LAA; red arrow: GCV

3D: Three-dimensional, Ex: Examination, Se: Series, DFOV: Display field of view, STND: Standard, Ph: Phase, BPM: Beats per minute, LCX: Left circumflex artery, RCA: Right coronary artery, LAD: Left anterior descending artery, Diag: Diagonal branch, CT: Computed tomography, LAA: left atrial appendage, GCV: Great cardiac vein

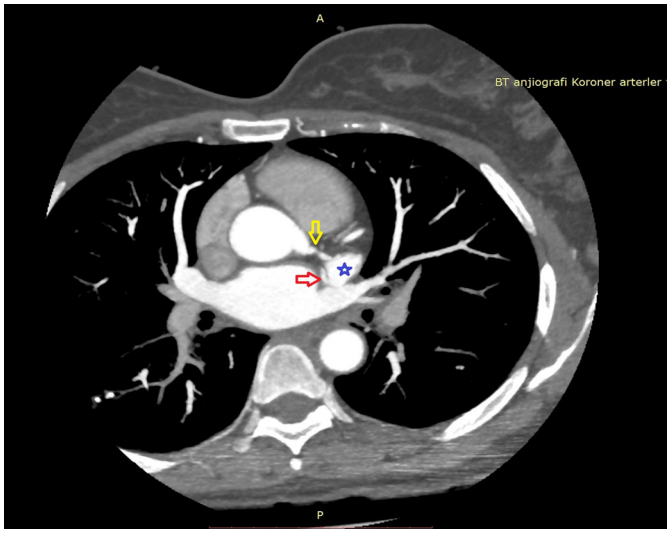

Figure 9. Case 2. Axial CT image. Blue star: LAA; red arrow: GCV; yellow arrow: left main coronary artery

CT: Computed tomography, LAA: left atrial appendage, GCV: Great cardiac vein

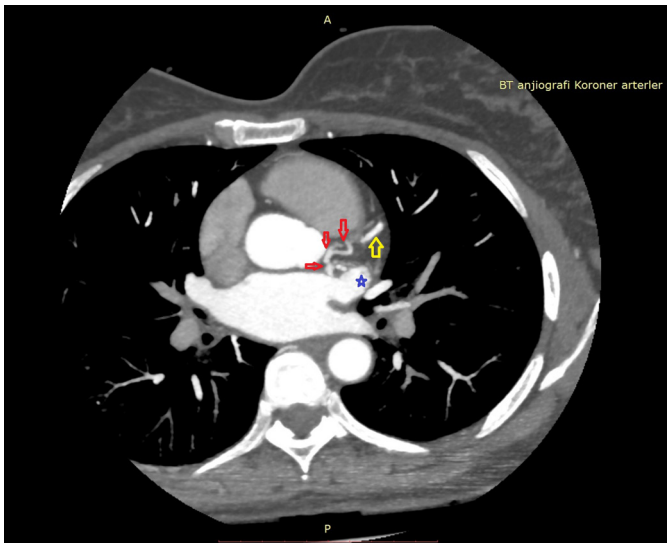

Figure 10. Case 2. Axial CT image, consecutive section following **Figure 9**. Blue star: LAA; red arrows: GCV; yellow arrow: LAD

CT: Computed tomography, LAA: left atrial appendage, GCV: Great cardiac vein, LAD: Left anterior descending artery

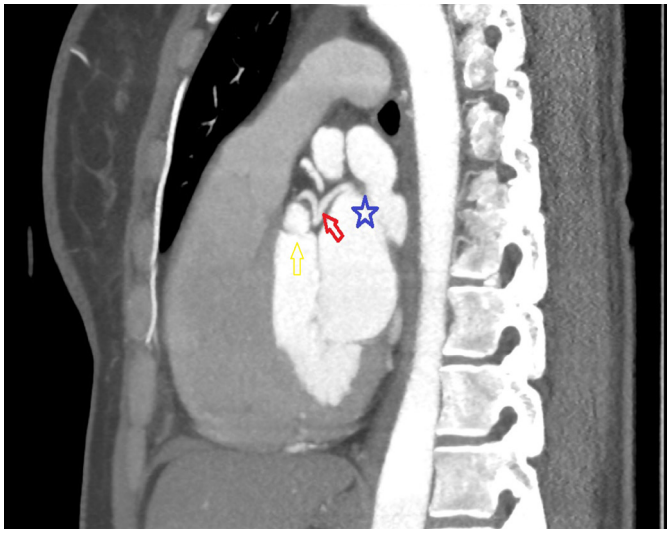

Figure 11. Case 2. Sagittal MIP CT image. Blue star: LAA; red arrow: GCV; yellow arrow: left ventricular outflow tract

MIP: Maximum intensity projection, CT: Computed tomography, LAA: left atrial appendage, GCV: Great cardiac vein

connections of the vessel with the standard venous tributaries were preserved.

A thin-walled CS, approximately 1.8 mm in diameter, was present in the posterior atrioventricular groove, with no connection to the GCV.

Quantitative measurements: The GCV measured 1.8 mm in the left atrioventricular groove and 2.4 mm immediately proximal to its termination in the LAA, and the anomalous course was 45 mm long. The LAA was of chicken-wing morphology, with maximum dimensions (AP×ML) of 12×29 mm.[15]

Clinical course and follow-up: The 2 mm secundum ASD was haemodynamically insignificant and required no intervention. As in Case 1, the patient was referred to cardiology and placed under clinical surveillance. At one year she reported no recurrent chest pain or dyspnoea, and no atrial arrhythmia or thromboembolic event had been documented. Follow-up beyond one year was not available in either patient and is acknowledged as a limitation.

DISCUSSION

Anomalous drainage of the GCV into the LAA is one of the least frequently reported coronary venous anomalies.[10-12] Yamamoto et al.[11] described a patient in whom the GCV drained into the left atrium and in whom atrial fibrillation of otherwise unexplained aetiology was present; Abazid et al.[10] reported anomalous coronary venous drainage into the left atrium detected on non-invasive imaging; and Aryal et al.[12] reported a bridging vein between the CS and the left atrium. In all of these reports the patients were adults in whom the anomaly was an incidental finding during imaging or electrophysiological study, and no consistent demographic pattern or syndromic association has emerged.

Differential Diagnosis

When a vascular structure is seen to connect with the LAA on CCTA, several entities must be excluded before a coronary venous drainage anomaly is diagnosed.

A PLSVC descends vertically anterior to the left pulmonary hilum, lateral to the aortic arch and the left pulmonary artery, and characteristically drains into a markedly dilated CS; it does not follow the atrioventricular groove and, after left-arm injection, opacifies before the right-sided structures. In both of our patients the anomalous vessel arose within the coronary venous territory, followed the atrioventricular groove and was not associated with a dilated CS.[1,2]

A left-sided or accessory pulmonary vein may lie close to the LAA and be mistaken for an anomalous vessel on volume-rendered images. Pulmonary veins can be traced peripherally into the lung parenchyma, opacify synchronously with the left atrium during pulmonary venous return and have no epicardial course within the atrioventricular groove, whereas the vessels described here could be followed along the atrioventricular groove and the course of the LAD and LCx arteries.[16]

A coronary arterial fistula arises from a coronary artery, enhances identically in timing and density to the coronary arteries, is typically dilated and tortuous, and drains into a low-pressure structure such as the right atrium, right ventricle or pulmonary artery. In our patients the anomalous vessel had no arterial origin and enhanced later and less densely than the coronary arteries, consistent with a venous structure.[17]

An arteriovenous malformation consists of a nidus of dysplastic vessels with a feeding artery and an enlarged draining vein, rather than a single, discretely traceable vessel following the course of a named cardiac vein. Neither a nidus nor early venous filling was present in either patient.[1]

Comparison of the Two Cases

The two patients shared several features: both were women in their early fifties, both were referred for CCTA because of chest pain rather than for any suspicion of a venous anomaly, neither had obstructive coronary artery disease or impaired systolic function, and in both the GCV terminated in the LAA and the anomaly remained clinically silent at one year. This concordance of gender and age is most probably a chance finding, given the very small numbers involved.

The cases differed principally in the course of the anomalous segment. In Case 1 the GCV followed an essentially normal epicardial route-along the anterior interventricular sulcus and then the left atrioventricular groove-and deviated only at its termination, so that the anomaly amounted to a distal misconnection of an otherwise normally coursing vein. In Case 2 the vessel was markedly tortuous and took an atypical route between the ascending aorta and the roof of the left atrium before reaching the LAA, so that both its course and its termination were abnormal. The incidental findings also differed: superficial myocardial bridging of the LAD in Case 1 and a small secundum ASD in Case 2.

Potential Clinical Implications

The clinical consequences of this anomaly remain theoretical, since no published case-including the two reported here-provides outcome data.

The LAA is the intracardiac structure most prone to thrombus formation, particularly in the presence of atrial fibrillation, because of its trabeculated structure and low flow velocities.[18] It has therefore been suggested that additional inflow from an anomalously draining vein might alter local flow conditions and, in theory, favour stasis and thrombus formation; this remains a hypothesis, and neither of our patients suffered a thromboembolic event during one year of follow-up.[10,18]

A relevant electrical connection between the LAA and the GCV has been demonstrated during catheter ablation of atrial fibrillation,[19] and atrial fibrillation of unexplained aetiology has been reported in a patient with anomalous GCV drainage into the left atrium.[11] Whether an anomalous GCV-LAA connection is itself arrhythmogenic cannot be determined from isolated reports, and neither of our patients had a documented atrial arrhythmia.

The implication we regard as clinically actionable is procedural: the anomaly should be recognised when planning LAA-targeted interventions, transseptal approaches and coronary sinus-related procedures.[2,4,5]

Incidental Findings

Case 1 also demonstrated myocardial bridging of the LAD. Although its length exceeded the 25 mm threshold associated with haemodynamic significance, an intramyocardial depth of less than 2 mm places the lesion in the superficial category, which carries low clinical risk.[20] No relationship between an interatrial defect, such as the small secundum ASD seen in Case 2, and anomalous GCV drainage has been described.[9] Neither finding has been reported in association with GCV-LAA drainage in the published cases.

Role of CCTA

As computed tomography technology has advanced, coronary venous anomalies have been reported with increasing frequency in the imaging literature.[2] CCTA is the preferred non-invasive imaging modality for recognising these anomalies and mapping them in three dimensions, owing to its near-isotropic high spatial resolution and to post-processing capabilities including cMPR, MIP and VR.[2,21]

This report is confined to two patients, and no inference regarding prevalence or risk can be drawn from it. The diagnosis rested on CCTA alone: neither patient underwent invasive venography, cardiac magnetic resonance imaging or electrophysiological study to confirm the connection or to establish the direction of flow. Because acquisition was optimised for the arterial phase, venous opacification was suboptimal and distal venous segments were incompletely opacified in Case 2; the total length of the GCV could therefore not be established with confidence, and the reported venous dimensions and lengths refer to the segments in which luminal contrast filling was demonstrable. CCTA also provides no information on the direction or volume of flow. Finally, follow-up was limited to one year and was based on outpatient cardiology review rather than on systematic rhythm monitoring; the longer-term follow-up required to assess thromboembolic and arrhythmic risk was not available.

CONCLUSION

In both of our patients this rare anomaly was clinically silent and required no intervention.

The individual venous trajectory should be described whenever the LAA or the coronary venous system is a procedural target. Rather than presuming an increased thromboembolic or arrhythmic risk, which the available evidence does not support, we suggest that the finding be reported descriptively and communicated to the referring cardiologist or electrophysiologist. Systematic attention to coronary venous anatomy in routine CCTA reporting, together with the accumulation of cases with adequate follow-up, offers the most realistic route to establishing the true prevalence and clinical relevance of this anomaly.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patients included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

This case report did not receive any financial support.

Author Contributions

Concept: MAK, AÖ; Design: MAK; Control: AÖ; Data Collection and/or Processing: MAK; Analysis and/or Interpretation: MAK, AÖ; Literature Review: MAK; Article Writing: MAK; Final Approval of the Manuscript: All Authors.

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