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ORIGINAL ARTICLE

Electrocardiographic characteristics in the patients with a persistent left superior vena cava

Kanako Ito-Hagiwara[1] · Yu-ki Iwasaki[1] · Meiso Hayashi[1] · Yujin Maru[1] · Yuhi Fujimoto[1] · Eiichiro Oka[1] · Kenta Takahashi[1] · Hiroshi Hayashi[1] · Teppei Yamamoto[1] · Kenji Yodogawa[1] · Yasushi Miyauchi[2] · Wataru Shimizu[1]

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Abstract

A persistent left superior vena cava (PLSVC) is a congenital venous abnormality and is usually asymptomatic and does not cause hemodynamic disturbances. Therefore, it is difficult to identify it by routine examinations in clinical practice. This study aimed to elucidate the electrocardiographic characteristics for the prediction of a PLSVC. Twelve patients (9 males, 56.2 ± 18.3 years) who were diagnosed with a PLSVC were enrolled. The electrocardiographic parameters, including the P-wave duration, axis, and morphology of the P waves, were automatically measured and compared to 150 controls (77 males, 57.3 ± 14.6 years). There were no significant differences in the P-wave duration. Negative or positive/negative P waves in lead III predicted a PLSVC with a sensitivity of 100% and specificity of 81%. The P-wave axis in PLSVC exhibited a significant leftward deviation as compared to the controls (14.8 ± 21.1 vs. 54.0 ± 17.4°, $p < 0.001$). A receiver operating characteristic curve analysis of the P-wave axis for predicting a PLSVC exhibited an area under curve of 0.93 [CI 95% (0.87–0.98), $p < 0.001$), and identified a P-wave axis of less than 37.5° to have a 92% sensitivity and 83% specificity in predicting a PLSVC. A negative or positive/negative P-wave morphology in lead III was a useful finding for suggesting the presence of a PLSVC.

Keywords Persistent left superior vena cava · P-wave morphology · P-wave axis · Electrocardiogram

Abbreviations

AF	Atrial fibrillation
CT	Computed tomography
CS	Coronary sinus
ECG	Electrocardiogram
SVC	Superior vena cava
TEE	Transesophageal echocardiography
TTE	Transthoracic echocardiography
PLSVC	Persistent left superior vena cava

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✉ Yu-ki Iwasaki
iwasaki@nms.ac.jp

1 Department of Cardiovascular Medicine, Nippon Medical School, 1-1-5 Sendagi, Bunkyo-ku, Tokyo 113-8603, Japan

2 Department of Cardiovascular Medicine, Nippon Medical School Chiba Hokusoh Hospital, Chiba, Japan

Introduction

A persistent left superior vena cava (PLSVC) is one of the common congenital venous abnormalities and affects 0.3–0.5% of the general population [1–3] and up to 10% of the patients with other documented congenital heart abnormalities [4]. A PLSVC is usually asymptomatic and does not cause any hemodynamic disturbances. Clinical and practical issues, however, may arise in cases undergoing a right-sided cardiac catheterization, catheter ablation, and pacemaker or cardioverter defibrillator implantations [5–10]. In addition, a PLSVC might be accidentally identified during cardiac examinations and operations [2]. It is preferable to discover the presence of a PLSVC prior to any catheter intervention or operation, because the venous anomaly is often associated with technical difficulties, which might increase the procedural time, radiation exposure time, and amount of contrast medium. A PLSVC can be diagnosed by transesophageal echocardiography (TEE) and chest computed tomography (CT), but these are not routinely performed in clinical practice [3, 11–13]. It would be quite useful if we could predict the presence of a PLSVC by the 12-lead electrocardiogram (ECG), which is the most common non-invasive cardiac examination, before any invasive examinations or operations. To date, little evidence has been reported regarding the ECG morphologies in patients with a PLSVC. The purpose of the present study was to elucidate the electrocardiographic characteristics for the prediction of a PLSVC.

Methods

Study population

The medical records were retrospectively reviewed in the 12 patients with a PLSVC diagnosed by TEE and/or chest CT at the Nippon Medical School Teaching Hospital from 2012 to 2017 (PLSVC group). One hundred and fifty consecutive patients who visited the department of cardiology for various chest symptoms such as palpitations or chest pain from 2015 to 2016 were selected as the control patients (control group). The patients who had a diagnosis of prior history of myocardial infarction, hypertrophic cardiomyopathy, dilated cardiomyopathy, or severe valvular disease were excluded from this study. The patients with regular negative P waves in lead II suggestive of a coronary sinus (CS) rhythm, which might have been a different atrial activation pattern than sinus rhythm, were also excluded [14]. This study was approved by ethics committee in Nippon Medical School Hospital.

Electrocardiographic analysis

All patients underwent a resting 12-lead ECG using an ECG-1550® digital recorder (Nihon Kohden Corporation, Tokyo Japan) with a sample rate of 500 samples/s and 1.25 µV/LSB. The ECG parameters were automatically measured by the ECG analyzing software (ECAPS12C®, Nihon Kohden Corporation, Tokyo, Japan). The ECG parameters included the P-wave morphology, P-wave duration, P-wave axis, PR interval, QRS duration, and heart rate. In cases with biphasic (positive/negative) P waves, the positive or negative P-wave amplitudes were calculated. When the P-wave amplitude was less than 20 µV, the P-wave morphology was defined as flat. The P-wave morphologies were classified into four groups consisting of positive, negative, biphasic (positive/negative), and flat groups according to the results of the automatic P-wave amplitude analysis. To determine the PLSVC prognostic value of the P-wave morphologies in the 12-lead ECG, the sensitivity and specificity were calculated according to the P-wave morphologies. ECG was obtained and analyzed before catheter ablation for the patient who scheduled for catheter ablation of atrial fibrillation.

Transthoracic echocardiographic analysis

A comprehensive transthoracic echocardiographic examination, including M-mode and Doppler echocardiography, were performed in all patients. The interventricular septal thickness, posterior left ventricular wall thickness, left atrial dimension, left ventricular end-diastolic dimension, and left ventricular end-systolic dimension were measured in the parasternal long-axis view. The left ventricular ejection fraction was also estimated by the long-axis view using the Teichholz method. Continuous wave Doppler of the tricuspid annulus was obtained from the apical 4-chamber view with a 5 mm sample volume. The cross-sectional area of the CS was measured in the long-axis view [15].

Statistical analysis

Statistical analyses were performed using independent sample tests. Continuous data were presented as the mean ± standard deviation, and categorical data were expressed as numbers and percentages. Differences between the two groups were tested using the Student's t test for continuous variables and Fisher's exact test for categorical variables. A p value of less than 0.05 was considered statistically significant. Receiver operator characteristic curves were generated to determine the sensitivity, specificity, and positive and negative predictive values of the top performing features using a selected liner discriminant classifier. All analyses were performed with the GraphPad Prism version 7.0 software (GraphPad Software, Inc., California).

Results

Baseline characteristics

In nine out of the 12 PLSVC patients, the CS drained into the right atrium; in seven patients, a left- and right-sided superior vena cava (SVC) was present, and in two patients, the right SVC was completely absent. For the remaining three patients, the classification was undetermined and contrast-enhanced CT and venography were not performed.

The clinical characteristics of the present study population are shown in Table 1. There was no difference between the PLSVC group and control group in terms of the age, gender, body mass index, presence of hypertension, diabetes mellitus, dyslipidemia, congestive heart failure, or cerebral infarctions. No difference was also seen in the serum electrolyte concentration such as the sodium and potassium levels between the two groups. Patients with a PLSVC had a significantly higher prevalence of atrial fibrillation (AF)

Table 1 Clinical characteristics of the PLSVC and control groups

	PLSVC ($N = 12$)	Control ($N = 150$)	p value
Age, years	55.8 ± 18.4	57.3 ± 14.6	0.74
Male, *n* (%)	9 (75%)	77 (51.3%)	0.14
Height, cm	167.4 ± 7.8	163.0 ± 8.5	0.149
Body weight, kg	68.7 ± 15.0	62.3 ± 12.8	0.16
Body mass index, kg/m^2	23.6 ± 4.1	23.4 ± 3.9	0.898
Hypertension, *n* (%)	7 (58.3%)	61 (40.6%)	0.2438
Diabetes mellitus, *n* (%)	2 (16.6%)	13 (8.7%)	0.307
Dyslipidemia, *n* (%)	2 (16.6%)	45 (37.5%)	0.511
Atrial fibrillation, *n* (%)	7 (58.3%)	28 (18.6%)	0.0044
Congestive heart failure, *n* (%)	1 (8.3%)	7 (4.7%)	0.465
Coronary artery disease, *n* (%)	0 (0%)	14 (9.3%)	0.602
Cerebral infarction, *n* (%)	0 (0%)	1 (0.7%)	1
Congenital heart disease, *n* (%)	1 (8.3%)	0 (0%)	0.0741
Brain natriuretic peptide, pg/ml	55.0 ± 38.0	57.5 ± 169.3	0.967
Na, mEq/l	141.4 ± 2.1	140.2 ± 2.1	0.0715
K, mEq/l	4.2 ± 0.4	4.2 ± 0.4	0.836
Blood urea nitrogen, mg/dl	14.0 ± 3.2	15.1 ± 5.2	0.51
Creatinine, mg/dl	0.7 ± 0.2	0.8 ± 0.4	0.677
Antiarrhythmic agents, *n* (%)	5 (41.7%)	10 (6.7%)	0.0019

PLSVC persistent left superior vena cava

compared to the control group (50% vs. 18.6%, $p = 0.0199$). Congenital heart disease, involving an atrial septal defect, was confirmed in one patient in the PLSVC group. The prevalence of a patent foramen ovale was higher in the PLSVC group than control group (16.7% vs. 0.7%, $p = 0.0146$).

Transthoracic echocardiographic findings in PLSVC

Table 2 shows the results of the transthoracic echocardiography (TTE) parameters. The interventricular septal thickness, left ventricular end-diastolic dimension, left ventricular end-systolic dimension, left atrial area, right atrial area, and tricuspid regurgitation pressure gradient were significantly greater in the PLSVC group than Control group. In addition, the area of the CS ostium was significantly larger in the PLSVC group than control group (2.1 ± 1.1 vs. 0.2 ± 0.1 cm^2, $p < 0.001$).

Electrocardiographic parameters and P-wave morphology

The electrocardiographic parameters between the two groups are presented in Table 2. There were no significant differences in the P-wave duration, PR interval, QRS duration, QT interval, and RR interval between the two groups.

A positive/negative or negative P wave in lead III predicted the presence of PLSVC with a sensitivity of 100% and a specificity of 81%. P-wave morphology patterns in control group and PLSVC groups are shown in Supplemental Table. The 12-lead ECG and P-wave patterns in the PLSVC group are demonstrated in Fig. 1.

The P-wave axis in the PLSVC group had a significant leftward deviation compared with that in the control group (14.8 ± 21.1 vs. 54.0 ± 17.4°, $p < 0.001$). The receiver operating characteristic curve revealed that the P-wave axis predicted a PLSVC with an area under curve of 0.93 (CI 95% 0.87–0.98, $p < 0.0001$), and identified that a P-wave axis of less than 37.5° had a 92% sensitivity and 83% specificity for the prediction of a PLSVC (Fig. 2).

Atrial activation pattern in the PLSVC

Figure 3 shows the angiography of the PLSVC before radiofrequency catheter ablation of AF in case 12. An enlarged CS and PLSVC were confirmed. The activation sequence of the intracardiac electrograms from the octapolar catheter located in the enlarged CS and ablation catheter located in the distal portion of the PLSVC exhibited a proximal to distal propagation of sinus rhythm. The activation of the electrograms in the PLSVC was almost identical to that of the terminal portion of the P wave. Angiography of the PLSVC in case 11 revealed the contraction of the PLSVC, indicating the presence of atrial working muscle within the vein (Supplemental Movie 1). Atrial activation mappings during sinus rhythm in case 12 using a three-dimensional electroanatomical mapping system (CARTO3, Biosense Webster, Diamond Bar, CA, USA; Ensite NavX system, St. Jude Medical, MN, USA) are shown in Fig. 4. The atrial activation initiated at

Table 2 Results of the transthoracic echocardiographic and electrocardiographic parameters of the PLSVC and control groups

	PLSVC ($N=12$)	Control ($N=150$)	p value
TTE parameters			
IVST, mm	9.8 ± 2.3	8.6 ± 1.8	0.0437
PWT, mm	8.8 ± 1.6	7.9 ± 1.4	0.0648
LAD, mm	32.8 ± 6.4.	32.8 ± 5.4	0.766
LVDd, mm	48.9 ± 5.0	46.0 ± 5.3	0.0486
LVDs, mm	30.5 ± 6.5	28.1 ± 5.0	0.0472
EF, %	67.1 ± 10.0	68.5 ± 8.6	0.271
LA area, cm^2	19.4 ± 7.4	15.9 ± 4.7	0.0184
RA area, cm^2	15.8 ± 4.3	12.6 ± 3.3	0.00474
AR, n (%)	2(16.7%)	22(14.7%)	0.693
MR, n (%)	1(8.3%)	18(12%)	1
TR, n (%)	5(41.7%)	30(20%)	0.136
CS ostium, cm^2	2.3 ± 1.4	0.2 ± 0.1	< 0.0001
ASD, n (%)	1(8.3%)	0(0%)	0.0741
ECG parameters			
P wave duration, ms	120.0 ± 23.8	114.9 ± 17.4	0.209
P axis, (°)	14.8 ± 21.2	54.0 ± 17.4	< 0.001
PR interval, ms	179.1 ± 49.1	166.9 ± 26.0	0.15
QRS width, ms	91.9 ± 11.1	95.1 ± 17.1	0.528
QT interval, ms	395.8 ± 29.5	394.3 ± 44.3	0.904
QTc	429.4 ± 23.2.	426.2 ± 23.9	0.655
RR interval, ms	878.3 ± 190.8	876.8 ± 165.5	0.975

PLSVC persistent left superior vena cava, *IVST* interventricular septal thickness, *PWT* posterior left ventricular wall thickness, *LAD* left atrial dimension, *LVDd* left ventricular end-diastolic dimension, *LVDs* left ventricular end-systolic dimension, *LVEF* left ventricular ejection fraction, *LA* left atrial, *RA* right atrial, *AR* aortic regurgitation, *MR* mitral regurgitation, *TR* tricuspid regurgitation, *TRPG* tricuspid regurgitation pressure gradient, *CS os* coronary sinus ostium, *ASD* atrial septal defect

the sinus node, which centrifugally propagated in the right atrium, and conducted to the left side through Bachman's bundle and the CS ostium. Atrial activation movies are shown in Supplemental Movie 2 and 3.

Discussion

Prevalence and diagnosis of a PLSVC

A PLSVC is the most common thoracic venous congenital malformation (0.3–0.5% in the general population [1, 2] and up to 10% in patients with other documented congenital heart abnormalities [4]), and normally obliterates in the 8th week of fetal life [16]. The common associated congenital heart abnormalities are a Fallot's tetralogy, Eisenmenger's complex, atrial septal defect, ventricular septal defect, transposition of the great vessels, and anomalous connections of the pulmonary veins [4]. In 8% of PLSVCs, there are no connections between the right atrium and enlarged CS, and a CS connecting into the left atrium can cause right-to-left shunting, resulting in cyanosis, and is commonly accompanied by atrial septal defects, arrhythmias, and other cardiac malformations. On the other hand, 92% of PLSVC connect into the right atrium through the orifice of an enlarged CS. Among them, almost 60% of cases have both a left- and right-sided SVC, and the others have the complete absence of a right SVC in the presence of a PLSVC [5, 16, 17]. The majority of those patients are asymptomatic and it is difficult to identify the presence of a PLSVC by a routine examination such as a chest X-ray or blood examination and physical examination.

A PLSVC is usually diagnosed by a chest CT, and echocardiography with both a TTE and TEE is also useful for the diagnosis of a PLSVC. If the TTE reveals CS dilatation, we should always consider the possibility of a PLSVC or a defect of the CS [18]. Conventionally, an injection of a bubble saline from the right cubital vein shows the contrast traveling from the SVC to the right atrium into the CS, suggesting the presence of a normal right SVC. If an injection of contrast from the left cubital vein shows the bubbles traveling from the CS into the right atrium, it suggests a PLSVC [11, 19].

Atrial activation pattern during sinus rhythm

Electrical signals from the sinus node located in the upper wall of the right atrium propagate to the right atrium and connect to the left atrium via Bachman's bundle and the atrial septum and CS. In this conduction system, Bachmann's bundle is also a branch of the anterior internodal tract that resides on the inner wall of the left atrium. It is a broad band of cardiac muscle that passes from the right atrium, between the SVC and ascending aorta [20]. Due to the sinus node being located adjacent to the SVC opening in the right atrium, the main vector of the atrial depolarization is pointed downward and leftward in the frontal plane, corresponding to a mean frontal plane P-wave axis of approximately 60°. Consequently, the atrial activation projects in the frontal plane as positive or upright P waves in leads I, II, and ${}_aV_F$, with some variability observed in leads ${}_aV_L$ and III, depending on the atrial geometry and exact orientation of the depolarization vector [21].

PLSVC architecture and P-wave morphology

A PLSVC is a malformation that arises because the left frontal basilar vein of the embryonic phase does not degenerate and does not close [22]. In the embryonic heart, bilateral pacemaking areas are present near the sinus horns and common cardinal veins [23]. Whereas the right side takes over

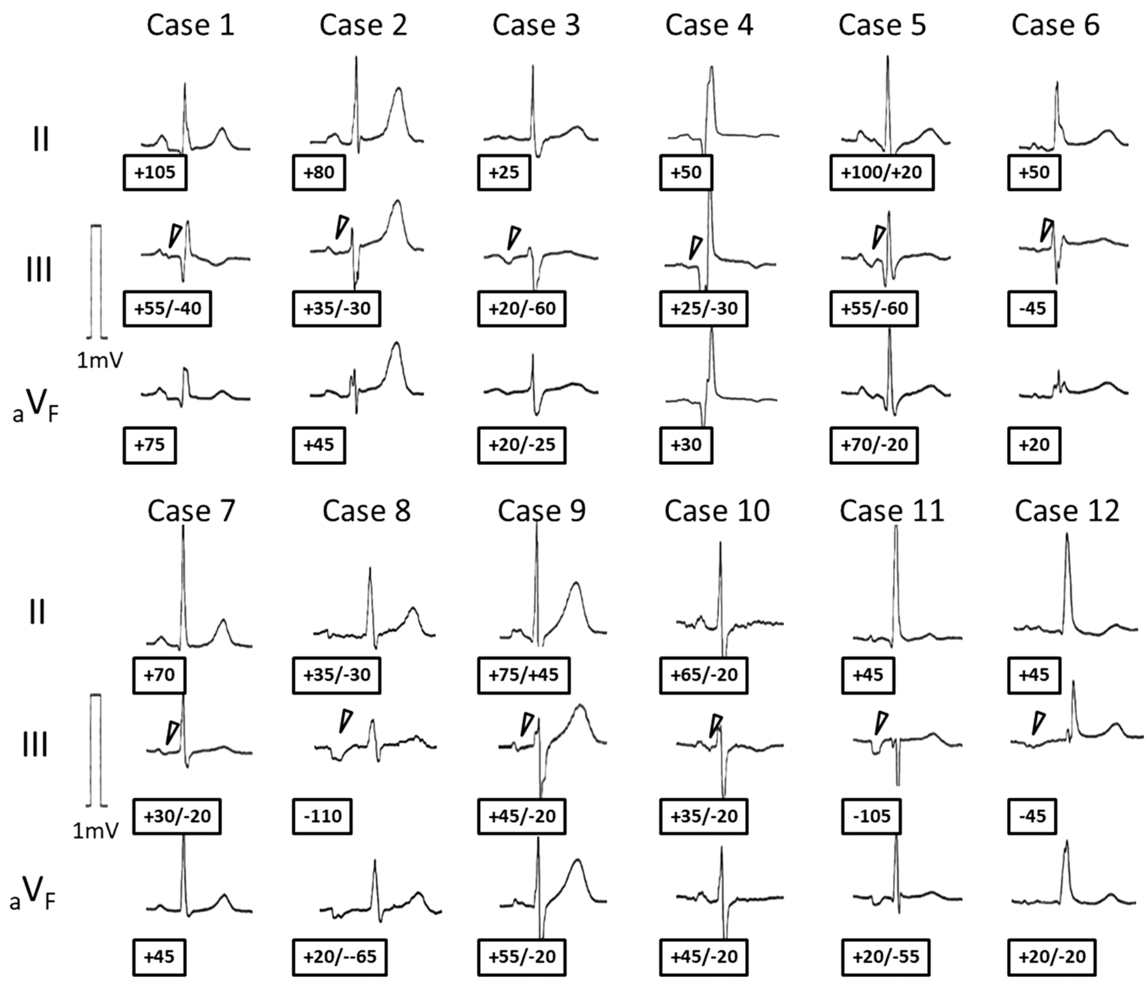

Fig. 1 ECG of lead II, III and $_aV_F$ in all 12 cases in the PLSVC group. In all cases, the P-wave morphology in lead III was either positive/negative or negative (white arrow). PLSVC indicates persistent left superior vena cava

the cardiac pacemaking function with the sinus node, the PLSVC may be associated with a continuing presence of pacemaker tissue and hence ectopic pacemaker activity [9]. The presence of electrical potentials within the PLSVC, consistent with the presence of muscle bundles, has been demonstrated with electroanatomical mapping [24]. Therefore, a PLSVC and enlarged CS contain much atrial muscle, which generates the atrial excitation affecting the P-wave morphology and vector. Indeed, the present study revealed that the atrial activation pattern during sinus rhythm using a 3D electrical–anatomical mapping system propagated from the enlarged CS ostium to the distal portion of the PLSVC. In addition, angiography of the PLSVC clearly showed the contraction of the PLSVC, implying the presence of a large amount of atrial working muscle cells in the PLSVC. In the present study, negative or positive/negative P waves in lead III in 100%, and positive P waves in lead $_aV_L$ in 83%, were confirmed in the PLSVC group. The direction of the excitation from the CS ostium to the distal portion of the PLSVC was opposite that of the vector of lead III and concordant with the vector of lead $_aV_L$, implying a better predictive value of a PLSVC in leads III and $_aV_L$, rather than II and $_aV_F$. These results suggested that some part of the terminal portion of the P wave associated with the PLSVC consisted of an enlarged CS and PLSVC excitation. An enlarged CS and the PLSVC excitation also caused a leftward P-wave axis deviation.

Biphasic P waves in the inferior leads in the absence of PLSVC

On the other hand, negative or positive/negative P waves in lead III were confirmed in 18.7% of the control group. There are several reasons for the low specificity of predicting a PLSVC from the P-wave morphology. It has been reported that Bayés syndrome is characterized by advanced interatrial block. It is a subclinical disease that manifests electrocardiographically as a prolonged P-wave duration of 120 ms or

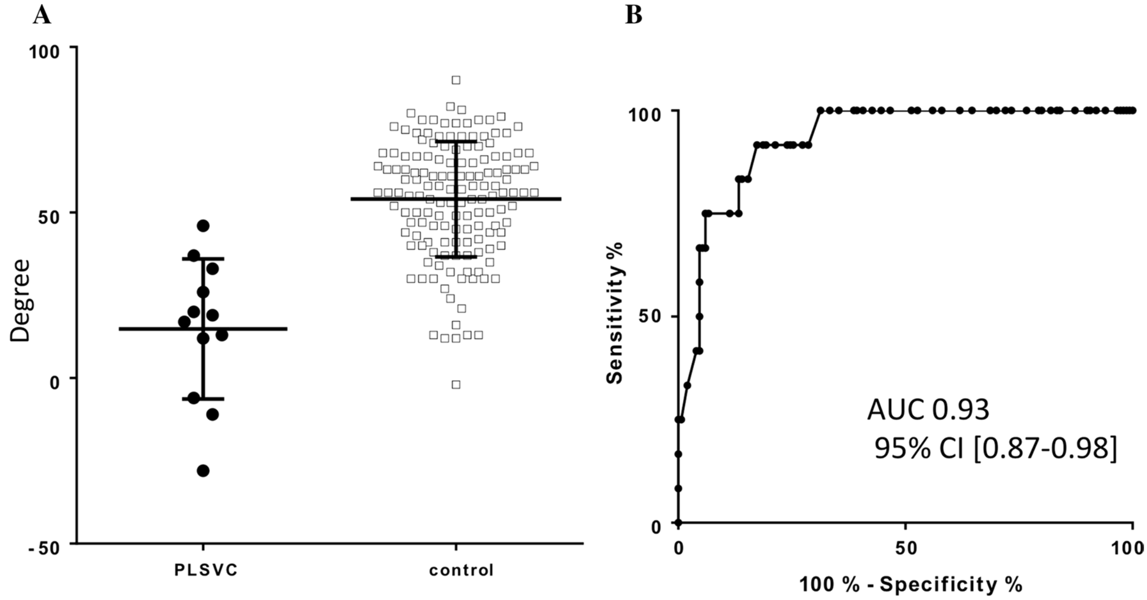

Fig. 2 **a** Box plots of the P-wave axis in the PLSVC and control groups. The P-wave axis in the PLSVC group exhibited a significant left deviation as compared to the control group (14.8 ± 21.1 vs. 54.0 ± 17.4°, $p < 0.001$). **b** Receiver operation characteristic curve for the prediction of a PLSVC. The P wave axis cut-off level was 37.5° (sensitivity; 92%, specificity; 83% and area under curve; 0.93). *PLSVC* persistent left superior vena cava, *AUC* area under curve, *CI* confidence interval

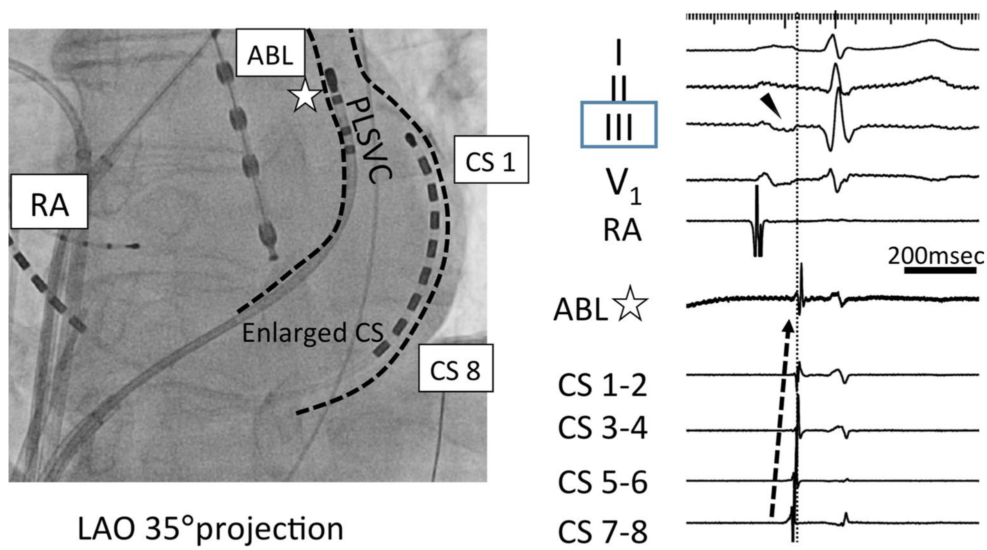

Fig. 3 Angiography during the radiofrequency catheter ablation of atrial fibrillation in case 12 showing an enlarged CS and PLSVC. *CS* coronary sinus, *PLSVC* persistent left superior vena cava, *RA* right atrium, *ABL* ablation catheter, *LAO* left anterior oblique

more with a biphasic morphology in the inferior leads [25]. A syndrome of an advanced interatrial conduction block due to conduction impairment in Bachmann's bundle results in a delayed and retrograde activation of the left atrium, and it is associated with a high incidence of atrial tachyarrhythmias, especially a particular and specific form of atypical atrial flutter or AF [26, 27]. In the presence of intra-atrial, conduction block (i.e., Bayés syndrome) might increase the false positive of the ECG criteria of P-wave morphology in lead III. An experimental animal study revealed that surgical lesions in the canine Bachman's bundle caused P-wave morphology changes characterized by biphasic P waves in the inferior leads [28]. Even in the patients with structurally normal hearts, atrial remodeling due to hypertension, aging, and AF might cause some conduction impairments in Bachmann's bundle. A recent study reported that conduction disorders of Bachmann's bundle are common and more pronounced in patients with AF. Therefore, those patients with the absence of a PLSVC, the P wave in the inferior leads might become negative, because the left atrium is propagated via the atrial septum and/or CS leading to a more caudo-cranial activation pattern of the left atrium [29–31].

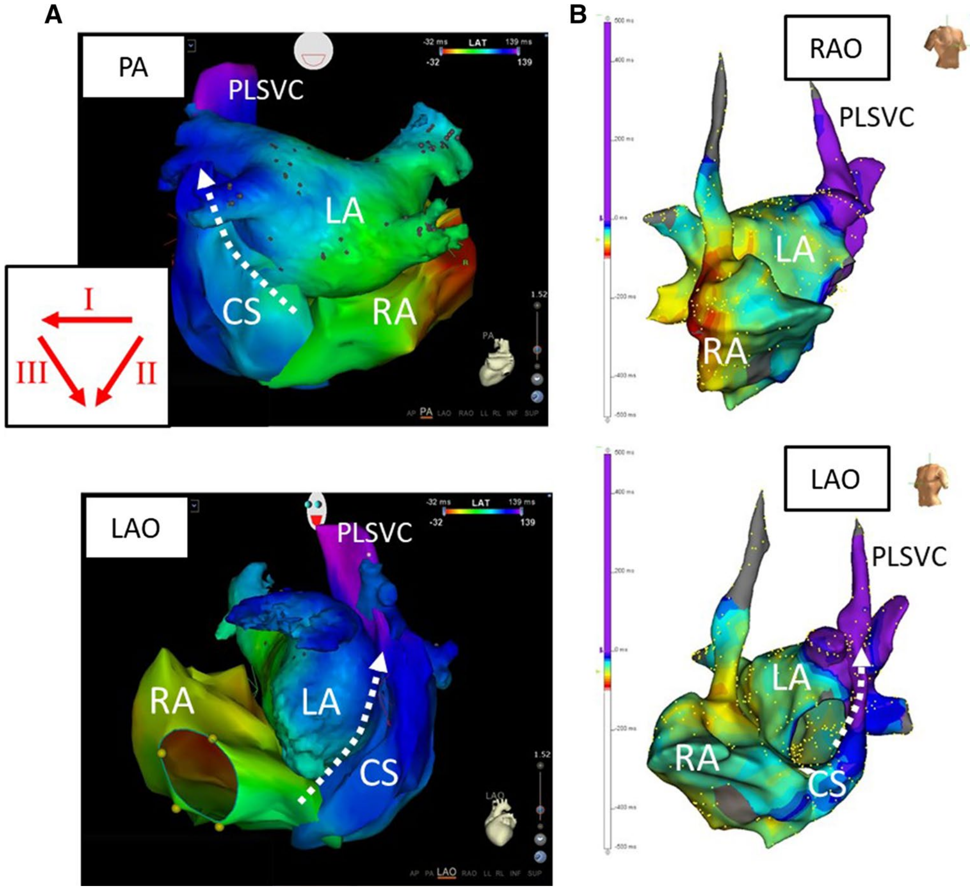

Fig. 4 **a** Atrial activation mapping in case 12. The atrial activation exhibited a proximal to distal propagation in the enlarged CS and PLSVC (white dash allow). The atrial activation was opposite direction to the lead III vector. **b** Atrial activation mapping in case 11. The propagation map in the posterior anterior view revealed a propagation in the left-cranial direction (white dash allows). *CS* coronary sinus, *PLSVC* persistent left superior vena cava, *RA* right atrium, *LA* left atrium, *PA* posteroanterior, *RAO* right anterior oblique, *LAO* left anterior oblique

Clinical implications

There are clinical advantages to the prediction of a PLSVC prior to a device implantation, catheter ablation, or cardiac catheterization in terms of reducing the radiation exposure, procedural time, and prevention of complications associated with the procedure. When the ECG exhibits negative or positive/negative P waves in lead III or leftward deviation of the P-wave axis during a routine examination in patients who are scheduled for such operations or examinations, a TTE, TEE, and chest CT should be performed to further investigate the presence or the absence of a PLSVC.

Limitations

In this study, there were several limitations. First, false positive of PLSVC diagnosis by P-wave ECG criteria in lead III was not few (≈ 18.7%). The patients having intra-atrial conduction block might increase the false positive. In those cases, transthoracic echocardiography will be needed to exclude the presence of PLSVC. In addition, we might not completely exclude the presence of a PLSVC in the control group. However, TTE was performed in all patients in the control group. There was no CS dilatation in the parasternal view implying the absence of a PLSVC [15]. Second, we chose the control group from the patients who were referred to the department of cardiology because of chest symptoms, including chest pain and palpitations. Indeed, the prevalence of AF was high in the control group. AF might cause an interatrial conduction delay affecting the P-wave morphology. In this study, the patients who had structural heart disease such as prior history of myocardial infarction and cardiomyopathy were excluded. These pathophysiological conditions might affect the P-wave morphology. The patient characteristics in the control group might have differed from those of the general population. Third, the P-wave morphology was difficult to classify in some cases. However, the P-wave morphology data were chosen from the ECG parameters, which were automatically measured by the computer software. There were no intra- or inter-observer differences for the evaluation of the P wave by this method.

Conclusion

A negative or positive/negative P-wave morphology in lead III was a useful finding for the prediction of the presence of a PLSVC.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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