Coexistence of Left Sided Atrioventricular Accessory Pathways with a Common Inferior Pulmonary Vein Ostium

Running title: Ihara et al.; Accessory pathway and Pulmonary vein anomaly

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Abstract:

Background - As the technique for radiofrequency catheter ablation for atrial fibrillation (AF) has progressed, so has our knowledge of both normal and abnormal anatomy of the left atrium and pulmonary veins (PV). We treated several AF patients with accessory conduction pathways (ACP) who were also found to have a common ostium of inferior PVs (CIPV), a relatively rare PV anomaly. No relation between ACP and PV anomalies has ever been reported, and the aim of our study was to study this association.

Methods and Results - This study included 137 consecutive patients (104 men, mean age 60±9 years) who underwent AF ablation for paroxysmal and persistent AF at our institution, from March 2009 to August 2010. We analyzed coexisting supraventricular tachycardias (SVT) and left atrium and PV morphology by multi-detector row CT. Thirty-eight of 137 patients (27.7 %) were found to have some PV anomaly, consisting of 13 with a common trunk of left PV, 19 with right additional PV, 3 with a common trunk of right PV, 3 with CIPV. Thirty-one patients (22.6 %) had SVT. They were: 26 atrial flutter, 4 WPW syndrome, 3 atrioventricular nodal reentrant tachycardia. The prevalence of a coexisting ACP was significantly higher in patients with CIPV than those without CIPV (3/3 (100 %) vs 1/134 (0.7%); p<0.0001). All ACPs with CIPV were located in the left side. The other SVTs were not associated with any PV anomalies.

Conclusions - There is a possible association between CIPV and left-sided ACP in AF patients. This suggests that there is a likelihood of developmental association between them.

Key words: common ostium of the inferior pulmonary veins, accessory pathway, atrial fibrillation, pulmonary vein, computed tomography
The pulmonary veins (PVs) have been demonstrated to be an important source of atrial ectopy initiating paroxysmal atrial fibrillation (PAF)\(^1\), and is the reason electrical isolation of the PVs by radiofrequency catheter ablation (RFCA) is highly effective for treatment of PAF\(^2,3\). Studies show that atrial ectopy in the PVs are associated with structural anomalies of the PVs\(^4-6\), and both multidetector row computed tomography (MDCT) and magnetic resonance imaging (MRI) can provide precise localization of such structures and the left atrium (LA), guiding RFCA\(^7,8\). Common ostium of the inferior PVs (CIPV) is a very rare anomaly of the PV that has been shown to be an important trigger of PAF\(^9\). However, an association between CIPV and other supraventricular tachycardias (SVT) has never been reported. Meanwhile, the atrioventricular accessory conduction pathway (ACP) is a common anatomical anomaly that causes atrioventricular reentrant tachycardia, but is rarely accompanied by structural cardiac abnormalities, except for those that are age-related\(^10\). We recently treated three patients with PAF who were found to have CIPV and coexisting ACP. The purpose of this study is to analyze the relationship between CIPV and ACP, and their characteristics.

**Methods**

This study included 137 consecutive patients who underwent AF ablation for drug-resistant paroxysmal and persistent atrial fibrillation at our institution from March 2009 to August 2010 and who underwent MDCT before the procedure. None had a history of prior LA ablation.

**MDCT imaging of the LA**

All patients underwent MDCT imaging at our institution, as is our standard
procedure prior to PVI. This involved non-gated contrast enhanced CT of the chest with a 64-detector helical CT scanner (Aquillon 64, Toshiba Medical Systems Co., Tokyo, Japan) using 64×0.5 mm collimation and a table feed of 45.0 mm/revolution, a pitch of 0.703, 120 kV, 400 mA, and 0.5 second rotation. All studies were performed with approximately 100 ml of 370 mg/ml Iopamidol (Iopamiron 370, Bayer Schering Pharma, Berlin, Germany) administered at a rate of 3.3 ml/s. The scanning delay was set with automated bolus-tracking technology (Real Prep technique, Toshiba Medical Systems Co.). As soon as the single density level in the LA increased to 200 Hounsfield units (HU) over baseline, the patient was instructed to take a deep breath and hold it. A processing workstation (Ziostation system610, Ziosoft Inc, Tokyo, Japan) allowed 3-dimensional viewing of volume-rendering images, virtual endoscopic images, and reformatted cross-sectional images in discretionary directions. The MDCT data sets thus obtained were exported to the CARTO electro-anatomical mapping system (CARTO XP, Biosense Webster, Inc., Diamond Bar, CA, USA) using CartoMerge image integration software (CARTO Merge Image Integration Module, Biosense Webster, Inc.).

**Electrophysiological Study and Catheter Ablation**

All antiarrhythmic drugs were withdrawn five half-lives before ablation; none of the patients were taking amiodarone. Warfarin was not discontinued for the procedure if the patient's CHADS2 score was greater than 1. PT-INR was controlled at 2.0-3.0. All patients underwent transesophageal echocardiography to rule out intracardiac thrombi 1 day before the procedure. All patients gave written informed consent for the electrophysiological (EP) study and RFCA. Anticoagulation was achieved with heparin (initial bolus of 5,000 IU intravenously, maintenance dose 1,000 IU/hour,
except for patients on warfarin who received an initial bolus of 3,000 IU, maintenance
dose 600 IU/hour). Analgesia was maintained with pentazocine (15 mg, bolus injection)
and propofol (10-20 mg/hour).

The basic sequence of the EP procedure was as follows. After transseptal puncture,
we performed left atriography, and placed three LASSO catheters (Biosense Webster,
Inc.) into the left superior (LSPV), left inferior (LIPV), and right superior (RSPV)
pulmonary veins. As needed, one Lasso catheter was moved to the right inferior PV
(RIPV) and isoproterenol was infused up to 4 μg/min. When spontaneous PV firing
was not observed even with isoproterenol, atrial fibrillation was induced by atrial burst
pacing from the electrode at the coronary sinus (CS) and terminated by intracardiac
defibrillation (5-10 J). After identification of the arrhythmogenic PV, registration of
the MDCT image prepared before the procedure was performed with a 7 F NaviStar
Thermo-Cool catheter (Biosense Webster, Inc.) and the CARTO system. Bilateral
extensive encircling ipsilateral PV isolation (EEPVI) was accomplished with the
double LASSO technique for all patients except those found not to have any
arrhythmogenic PV.

If spontaneous firing was observed in the superior vena cava (SVC), SVC isolation at
the level of the SVC ostium was performed after SVC venography. Patients with a
history of atrial flutter also underwent ablation of the cavotricuspid isthmus.

After all RFCA applications, we performed an EP study including infusion of
isoproterenol up to 20 μg/min to confirm absence of inducibility of any
supraventricular tachycardia except atrial fibrillation or atrial tachycardia. Lastly, we
checked for dormant PV-LA conduction with a 40 mg bolus infusion of adenosine
triphosphate (ATP), to detect presence of any coexisting ACP.
All RFCA was performed under guidance of the CARTO Merge system. Radiofrequency energy was delivered for 20 to 30 seconds at each ablation site with a maximal temperature of 45°C, a power output of 25-35 W, and an irrigating rate of 17-30 mL/min. When ablating the posterior LA wall, the RFCA application was limited to 25W and duration of 20 seconds to reduce risk of thermal damage to the esophagus.

**Definitions**

The morphology and branching pattern of PVs were analyzed using the MDCT images. In the present study, we defined additional PVs as extra veins with independent atrio-venous junctions from the superior and inferior PVs [11,12]. If the distance between the virtual border of the LA and the bifurcation of left or right PVs was 15 mm or greater, the left or right ostium respectively was defined as being common [11,12]. CIPV was defined as bilateral inferior PVs having a common ostium which could be recognized distinctly in the endocardial view. All images were analyzed by two independent observers unaware of the clinical data.

**Statistical Analysis**

Data are reported as mean±SD. Fisher’s exact test was utilized to compare categorical variables. Statistical analysis was performed using SPSS version 18.0. A p value of <0.05 was considered statistically significant.

**Results**

Of 137 consecutive patients (104 men, mean age 60±9 years, paroxysmal/persistent atrial fibrillation; 118/19 pts), 38 patients (27.7 %) had some thoracic vein anomalies in their MDCT image, consisting of 13 (9.5 %) with common trunk of the left PV, 19
(13.9 \%) with right additional PV, 3 (2.2 \%) with common trunk of the right PV, 3 (2.2 \%) with CIPV, 1 (0.7 \%) with persistent left superior vena cava, and 1 (0.7 \%) with interruption of the inferior vena cava with azygos continuation (2 patients had 2 PV anomalies).

Thirty-one patients (22.6 \%) also had SVTs other than atrial fibrillation or atrial tachycardia. They were: 26 atrial flutter (19.0 \%), 4 WPW syndrome (2.9 \%), 3 atrioventricular nodal reentrant tachycardia (2.2 \%) (2 patients had 2 of these arrhythmias). It is particularly worth noting that all three patients with CIPV had a coexisting ACP. The prevalence of a coexisting ACP was significantly higher in the patients with CIPV compared to those without CIPV (3/3 (100 \%) vs 1/134 (0.7 \%); p<0.0001, Table 1). The other SVTs were not associated with any PV anomalies.

The characteristics of these 3 patients are shown in Table 2, and we present their case reports below.

**Case 1**

A 45-year-old man suffering from paroxysmal narrow QRS regular tachycardia, not atrial fibrillation, was referred to our institution for RFCA. A delta wave had been found in his 12-lead surface electrocardiogram(ECG) at the age of 36. He was on medications for hypertension, but had no past history nor family history of other cardiac disease. His echocardiogram showed normal cardiac function and no obvious structural heart disease. A delta wave was apparent on his admission ECG (figure 1-A). EP study revealed atrioventricular reentrant tachycardia complicated with PAF and we performed RFCA only for the left lateral ACP by the conventional ventricular approach. However, we found it necessary to apply RF energy in the CS to eliminate retrograde ACP conduction. When we were done ablating the ACP, retrograde and
antegrade ACP conduction were not observed even with ATP. Atrioventricular reentrant tachycardia could not be induced by programmed stimulation even with isoproterenol.

Some time later, the patient returned to the hospital complaining of palpitations and PAF was clinically documented for the first time. His PAF was drug-resistant, and we performed PV isolation after conducting MDCT to study LA and PV morphology. The MDCT showed dilation of the ostium of RSPV and CIPV (figure 2-A). Although delta waves were absent from his ECG at second admission, EP study revealed the recovery of retrograde conduction through the left lateral ACP and orthodromic atrioventricular reentrant tachycardia was easily induced by programmed stimulation. We unsuccessfully attempted RFCA of the ACP via an atrial approach (through transseptal puncture), and once again succeeded only after RFCA application in the CS. Ectopy initiating PAF frequently occurred from the CIPV (LIPV) and LSPV. We attempted PV isolation with "Box isolation" using the original method \(^\text{13}\) of the double Lasso technique under guidance of CARTO merge system, but this attempt was not successful. Therefore, we performed a "tri-circle ablation" \(^\text{9}\), with which we achieved PV isolation (figure 3-A).

Case 2

A 58-year-old man, who had undergone RFCA for overt WPW syndrome at another institution one year before, was referred to our institution for drug-resistant PAF. There were no delta waves on his admission ECG (figure 1-B). He had no other past history and no family history of heart disease. His echocardiogram showed normal cardiac function and no obvious structural heart disease. His MDCT image showed CIPV (figure 2-B). His EP study revealed that retrograde ACP conduction remained, but not antegrade ACP conduction, and orthodromic atrioventricular reentrant tachycardia was
easily induced. Spontaneous firing also occurred frequently from the CIPV (RIPV) and LSPV. We achieved ablation of the ACP by the transseptal atrial approach, and PV isolation for the CIPV was completed by a "Box isolation" (figure 3-B) with double Lasso technique under guidance of CARTO merge system. Complete isolation of the whole of the LA posterior wall and 4 PVs was confirmed by the absence of local electrical activity in them and the inability to capture outside the box during pacing in each of them.

**Case 3**

A 56-year-old man was referred to our institution for symptomatic drug-resistant PAF. His symptoms had always occurred with PAF without delta wave, and narrow regular tachycardia had never been documented. He had polycystic liver, for which he was receiving no specific therapy. He had no other past history and no family history of heart disease. His echocardiogram showed normal cardiac function and no obvious structural heart disease. We scheduled his PV isolation, and MDCT was performed before the procedure, which revealed CIPV and dilation of the RSPV ostium (figure 2-C).

A delta wave was observed on his ECG at admission (figure 1-C), and the patient was diagnosed with intermittent WPW syndrome for the first time. EP study suggested an ACP located in the left posterior area (figure 3-C), but no retrograde ACP conduction was observed, and antegrade ACP conduction was rarely observed (intermittent). Atrioventricular reentrant tachycardia was not inducible even with isoproterenol. We did not perform RFCA for ACP, because his ACP was considered a bystander, and because we couldn't determine its location due to its intermittency. This patient's PVs including the CIPV showed no ectopic activity. Instead, frequent
spontaneous firings initiating PAF were observed repetitively from the SVC. Therefore, we performed only SVC isolation. After SVC isolation, atrial fibrillation could not be induced with isoproterenol and atrial burst pacing.

**Follow-up after Catheter Ablation**

Over the mean 6 months of follow-up after the RFCA procedure, there were no obvious complications related to the procedure. All three cases remained free from atrial fibrillation and paroxysmal supraventricular tachycardia in the absence of antiarrhythmic drugs.

**Discussion**

The present study is the first to describe a possible association between a PV anomaly and ACP. The incidence of ACP detected electrocardiographically has been reported to be 0.1-3.1/1000 in people of all ages, and atrial flutter-fibrillation may be the presenting arrhythmia in 5-10% of patients with ACP\(^\text{10}\). The majority of patients with ACP have no heart disease\(^\text{10}\), but, some ACPs are associated with certain congenital heart abnormalities, particularly Ebstein's anomaly of the tricuspid valve\(^\text{14}\). Mitral valve prolapse, hypertrophic obstructive cardiomyopathy, and some genetic defects have been also reported to accompany ACP\(^\text{10,15,16}\). Recently, some reports attempted to demonstrate the incidence and characteristics of paroxysmal supraventricular tachycardia in atrial fibrillation patients. According to those reports, AVRT could be induced during electrophysiological study for PAF ablation in 1.7-1.9% of the patients\(^\text{17,18}\). Furthermore, successful catheter ablation of accessory pathways was found to prevent further recurrence of AF in 91% of patients in one study\(^\text{19}\). However, no reports have ever analyzed an association between a PV anomaly
Conversely, MDCT and MRI of the LA and PVs are now widely performed to assess atrial anatomy prior to procedures for atrial fibrillation\textsuperscript{7,8}, and physicians are becoming increasingly familiar with normal and abnormal anatomy of these structures. In previous reports, a common trunk of the left or right PV, the right additional PV were occasionally described\textsuperscript{4,6,8}, but CIPV was thought to be a very rare condition\textsuperscript{9,20-26} observed in 0.9-1.5 % of atrial fibrillation patients\textsuperscript{9,20}. Furthermore in those reports, the relation between CIPV and SVTs other than atrial fibrillation was not assessed, and some of those included patients with a history of prior RFCA application for LA which may have affected LA and PV morphology. Our study consisted entirely of patients who underwent first time LA ablation. Given the rareness of ACPs on their own, and the rareness of CIPVs on their own, the presentation of 3 patients having both ACP and CIPV triggered our interest.

Characteristics of the ACPs with CIPV

The patients with CIPV were all middle-aged men without any structural heart disease. Electrocardiographic delta waves were recorded in all three. All ACPs were located in the left side (figure 3), and there were no specific macroscopic structures around the ACP in their MDCT images. In the only WPW syndrome patient without CIPV, the ACP was located on the right side.

Some previous studies have demonstrated coronary vein anomalies related to accessory atroventricular pathways, especially the posteroseptal epicardial ACPs\textsuperscript{27-29}. In this study, all three patients underwent coronary venography during the procedure, but no anomalies of the coronary sinus were detected.

In case 1, the ablation of ACP failed with both a ventricular and atrial approach,
and RFCA application in the CS was needed to eliminate his ACP. In endocardial RFCA of ACPs, ablation failure may be due partly to the epicardial insertion of the ACP.27-30

Characteristics of CIPV

As in past reports9,20, the MDCT imaging (figure 4) revealed no structures around the common ostia external to the LA and PVs, that could possibly have obstructed development of the PV forcing CIPV to remain undifferentiated. The common trunks of the inferior PVs projected in a posterior direction, and were very close to the esophagus. It is very important to avoid ablating these areas, or to reduce RF energy to reduce the risk of damage to the esophagus.

Some PV anomalies have been associated with the initiation of atrial fibrillation4-6. In a past report, focal ectopy followed by atrial fibrillation was observed from 4 of 11 CIPVs9. In this study, 2 of 3 patients with CIPVs had spontaneous firing from the CIPV during their procedure session. CIPVs may play an important role in triggering atrial fibrillation, similar to other type of PV anomalies.

Development of PV and atrioventricular septation

Embryologically, the common PV joins to the primary atrial component of the heart and starts to bifurcate, dilate and, incorporate into the LA at a gestational age of around 30 days. By 7-8 weeks of gestation, contemporaneously with septation of the atriums and ventricles, the common PV separates into the RPV and LPV, and positions itself in the most inferior part of the LA, adjacent to the atroventricular groove and CS. At the age of 15 weeks, it is possible to identify four PV ostia draining into the LA.31 During this developmental process, incomplete PV incorporation can result in a unilateral common PV ostium, while extreme PV incorporation can lead to more than 4
The atrioventricular sulcus and cushions develop concurrently with PV development, and the continuity of atrial and ventricular muscle is interrupted gradually. At the end of the third month of gestation, the atrial myocardium and ventricular myocardium are almost completely separated in normal subjects. ACPs in WPW syndrome are thought to be derived from abnormal remnants of muscle fibers that provided direct continuity between atrial and ventricular myocardium. Normal regression of muscle fibers during atrioventricular septation may be inhibited by a molecular defect.

We speculate that the developmental abnormalities giving rise to CIPV and ACP may be related, perhaps by a molecular defect. We note that in the present study, the CIPV were in the inferior LA, and all of the ACP were in the posterior to lateral LA atrioventricular border, near the inferior LA. We think that it is of significant interest that developmentally at the time of common PV bifurcation, the common PV is adjacent to the atrioventricular groove, suggesting a possible common etiology. Furthermore, the other types of PV anomalies were not related to ACPs or other supraventricular tachycardias, suggesting that CIPV may belong to a different class of PV anomalies from the others, perhaps due to its proximity to the AV border.

**Clinical implications**

Some consideration is required in treating atrial fibrillation with CIPV. First, CIPV is a potential target vein of PV isolation. However, the standard ablation strategy of PV isolation, EEPVI, is very difficult in CIPV, especially the making of a longitudinal line on the LA posterior wall. Although one report recommends "tri-circle" ablation,
we consider Box isolation to be a preferable strategy for CIPV, because Box isolation does not need a transverse RFCA line to the posterior aspect of the LA, which is thought to be closer to the esophagus than other LA and PV morphologies\textsuperscript{13). Secondly, the association of CIPV with ACP should be considered. When you treat atrial fibrillation with CIPV, you should carefully investigate whether there is coexistence of ACP.

**Limitations**

There are several limitations to the study. First, it was a retrospective analysis with a small number of patients, and the study group consisted solely of patients who underwent atrial fibrillation ablation, i.e., it was a selected population and different from the general population. Second, this study did not include patients who underwent ablation only for WPW syndrome, because MDCT is not routinely performed for WPW patients at our institution. Therefore, we could not assess the prevalence of CIPV in WPW syndrome. A prospective and larger study is needed to investigate the precise relationship between CIPV and ACP.

**Conclusions**

We found a possible association between a unique pulmonary vein morphology, CIPV, and left-sided accessory pathway in atrial fibrillation patients. This suggests a developmental association between them.

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Conflict of Interest Disclosures: None.

References:


34. Lunel AA. Significance of annulus fibrosus of heart in relation to AV conduction and ventricular activation in cases of Wolff-Parkinson-White syndrome. Br Heart J. 1972; 34:1263-1271.
**Table 1.** The prevalence of Supraventricular tachycardia in each PV anomalies

<table>
<thead>
<tr>
<th></th>
<th>RAPV</th>
<th>CLPV</th>
<th>CRPV</th>
<th>CIPV</th>
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<tr>
<td>WPW n, (%)</td>
<td>0 (0)</td>
<td>4 (3.4)</td>
<td>0 (0)</td>
<td>3 (100)</td>
</tr>
<tr>
<td>AVNRT n, (%)</td>
<td>0 (0)</td>
<td>2 (1.7)</td>
<td>0 (0)</td>
<td>3 (100)</td>
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<tr>
<td>AFL n, (%)</td>
<td>3 (15.8)</td>
<td>23 (19.5)</td>
<td>2 (15.4)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

RAPV=Right additional pulmonary vein, CLPV= Common trunk of the left pulmonary vein, CRPV= Common trunk of the right pulmonary vein, CIPV= Common ostium of the inferior pulmonary veins, WPW=Wolf-Parkinson-White syndrome, AVNRT=Atrioventricular nodal reentlant tachycardia, AFL=Atrial Flutter

**Table 2.** Characteristics of the Patients with Common Ostium of the Inferior Pulmonary Veins

<table>
<thead>
<tr>
<th>Patients</th>
<th>Sex</th>
<th>Age</th>
<th>Complicating disease</th>
<th>Type of AF</th>
<th>AF trigger</th>
<th>Coexisting ACP</th>
<th>Type of WPW syndrome</th>
<th>ACP location</th>
<th>AVRT</th>
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<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>46</td>
<td>Hypertension</td>
<td>Paroxysmal</td>
<td>LIPV, LSPV</td>
<td>+</td>
<td>overt</td>
<td>Left lateral</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>58</td>
<td>None</td>
<td>Paroxysmal</td>
<td>RIPV, LSPV</td>
<td>+</td>
<td>overt</td>
<td>Left postero-lateral</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>56</td>
<td>Polycystic liver</td>
<td>Paroxysmal</td>
<td>SVC</td>
<td>+</td>
<td>intermittent</td>
<td>Left posterior</td>
<td>-</td>
</tr>
</tbody>
</table>

Figure Legends:

Figure 1. 12-lead resting electrocardiogram of three patients with common ostium of the inferior pulmonary veins. ECGs A, B, and C are from cases 1, 2, and 3, respectively, described in the text. Within each case, the two strips on the left are from before RFCA, and the two on the right are from after. Delta waves are visible in A and B before the RFCA but not after the ablation. The Case 3 patient had intermittent type WPW syndrome which was diagnosed for the first time at admission for catheter ablation of paroxysmal atrial fibrillation (C).

Figure 2. MDCT imaging of patients with common ostium of the inferior pulmonary veins (CIPV). Posterior images of the left atrium in patients with CIPV are shown. A, B, and C represent cases 1, 2, and 3, respectively, as described in the text. In A and C, dilation of the ostium of the right superior PV is also observed.

Figure 3. Anterior endoscopic view of the left atrium (LA) in patients with common ostium of the inferior pulmonary veins (CIPV) and their ablation site. The anterior endoscopic LA images of cases 1, 2, and 3 (as described in the text) produced by the CARTOmerge system are shown in A, B, and C, respectively. The CIPV can be seen from inside the LA. In each image, the RFCA application sites for PV isolation (Red dot) and the AP location (white dot and arrow) are shown, except that in case 3, a white dot is placed at the site where ACP was thought to exist, because ablation for the ACP was not performed. In case 1, tri-circle ablation (A) was performed for paroxysmal atrial fibrillation ablation, and in case 2, box isolation (B).
Figure 4. MDCT axial and sagittal view of the left atrium (LA) and common ostium of the inferior pulmonary veins (CIPV). The axial and sagittal views of the LA and CIPV in cases 1, 2, and 3 as described in the text are shown in A, B, and C, respectively. The common trunks of the inferior PVs projected in a posterior direction. The position of the esophagus is marked with white arrows to demonstrate its proximity to the CIPV. There were no macroscopic structures external to the LA that could have obstructed normal development of the PVs.
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