The following factors are in favor of this hypothesis: no ILS case among 42,000 infants < 2 months of age; normal pulmonary venous drainage; rariety of ILS in lobes other than the lower lobes (pulmonary ligament arteries do not exist in the upper lobes); and fewer ILS cases with other abnormalities when compared to extralobar sequestration (ELS) cases.2,3 But this hypothesis cannot explain patients with both ILS and ELS, patients with ILS who have gut fistulas, accompanying congenital malformations with an incidence of 14%, bilateral sequestrations of the same or different types, ILS patients with upper lobe localization, or cases of ILS in neonates.4–17 Besides this, it is clear in this case that there is no need for the acquired neovascularization (parasitization from aortic arteries) of sequestrated lung tissue that has already been supplied by an artery large enough and is unquestionably congenital in origin.

The wheel theory of Clements et al18 seems to be more acceptable in explaining the development of congenital bronchopulmonary malformations. According to Clements et al,19 ILS and ELS have similar origins. If the patient with ILS whose case was presented by Lewis and Tsou had been operated on and, thus, had had ILS histopathologically confirmed (eg, bronchial obstruction, occlusion of the pulmonary artery, or a type of pleural envelope lesion), collateral vessels originating from the aortic arch and reaching the lesion could have been claimed to be acquired originally because of neovascularization secondary to chronic infection. This case is congenital in origin because of the abnormal artery originating from the circumflex artery.

In addition, ELS is frequently localized in the left lower lobe, just between the lobe and the diaphragm in its own pleural envelope. The type of pleural envelope in the lesion (IL-EL) cannot be detected by CT scanning alone but requires histopathologic verification. The venous drainage in this case also has not been clearly identified. Pulmonary venous drainage is characteristic in patients with ILS. When the diagnosis is uncertain, surgery is the rule in sequestrations.13,14 Two other limitations of this case are the discordance with the acquired ILS hypothesis of Stocker and Makzak,2 in that arteries do not reach the lesion via the ligamentum pulmonale, and the lack of a bronchologic study. There are ILS cases with communication to the tracheobronchial tree or cases in which inflammatory changes occur in adjacent bronchi.19 In this case, the patient has a history of bronchitis of quite a long duration. Collateral arteries from the aortic arch to the lesion are not proof enough that the lesion was acquired. Besides, by means of collateral ventilation, the sequestrated portion of the lung in patients with ILS can be ventilated and infected.20 The above-mentioned sequential events do not necessarily mean that ILS is acquired as a result of infection.

I believe that this case of sequestration is congenital in origin, and, with respect to the above-mentioned reasons, it will not be

---

### Table 1—Usefulness of Different Parameters in the Separation of Transudates and Exudates*

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>PF cholinesterase</td>
<td>96</td>
<td>96.4</td>
<td>97.7</td>
<td>79.4</td>
<td>89.8</td>
</tr>
<tr>
<td>PF/S cholinesterase</td>
<td>96</td>
<td>96.4</td>
<td>97.9</td>
<td>93.1</td>
<td>96.2</td>
</tr>
<tr>
<td>Criteria of Light et al</td>
<td>98</td>
<td>82.2</td>
<td>90.7</td>
<td>95.4</td>
<td>92.4</td>
</tr>
</tbody>
</table>

*Values given as percentages. PPV = positive predictive value; NPV = negative predictive value.

---

### References


---

To the Editor:

We thank Dr. Sevim and colleagues for their interest in our article (July 1996).1 At present, we do not recommend the use of the pleural fluid to serum cholinesterase ratio (PF/S ChE) for the separation of transudates and exudates. Although this test has a similar accuracy to that of the criteria of Light et al, we found that a significant number of misclassified exudates with the PF/S ChE were malignant,2 and this fact is a very important limitation on its clinical use.

Eduardo Garcia-Pachon, MD
Hospital Vega Baja
Orihuela-Alicante, Spain
Isabel Padilla-Navas, MD
Hospital General Universitari d’Elx
Alicante, Spain

Correspondence to: Eduardo Garcia-Pachon, MD, Department of Internal Medicine, Hospital Vega Baja, E-03314 Orihuela-Alicante, Spain

---

### Intralobar Pulmonary Sequestration

To the Editor:

I read with interest the case of intralobar pulmonary sequestration (ILS) that appeared in the June 2000 issue of CHEST as a Roentgenogram of the Month.1

The development of ILS, one of the most interesting developmental pulmonary abnormalities, has not been elucidated yet. I do not agree with the comments of the authors on this case regarding the etiology as being both acquired and congenital in the same patient. The quite rare and large aberrant systemic artery originating from the left circumflex coronary artery and reaching the left lower lobe is proof enough that this case is congenital in origin. In the study of Stocker and Makzak,2 arteries from the thoracic aorta branching into the visceral pleura traversing the ligamentum pulmonale have been found to be anatomically present, and, depending only on that criterion, it was claimed that a chronically infected lesion obstructing a bronchus and occluding the pulmonary artery could cause acquired ILS by means of systemic arterial parasitization from aortic arteries traversing the ligamentum pulmonale.

The following factors are in favor of this hypothesis: no ILS case among 42,000 infants < 2 months of age; normal pulmonary venous drainage; rariety of ILS in lobes other than the lower lobes (pulmonary ligament arteries do not exist in the upper lobes); and fewer ILS cases with other abnormalities when compared to extralobar sequestration (ELS) cases.2,3 But this hypothesis cannot explain patients with both ILS and ELS, patients with ILS who have gut fistulas, accompanying congenital malformations with an incidence of 14%, bilateral sequestrations of the same or different types, ILS patients with upper lobe localization, or cases of ILS in neonates.4–17 Besides this, it is clear in this case that there is no need for the acquired neovascularization (parasitization from aortic arteries) of sequestrated lung tissue that has already been supplied by an artery large enough and is unquestionably congenital in origin.

The wheel theory of Clements et al18 seems to be more acceptable in explaining the development of congenital bronchopulmonary malformations. According to Clements et al,19 ILS and ELS have similar origins. If the patient with ILS whose case was presented by Lewis and Tsou had been operated on and, thus, had had ILS histopathologically confirmed (eg, bronchial obstruction, occlusion of the pulmonary artery, or a type of pleural envelope lesion), collateral vessels originating from the aortic arch and reaching the lesion could have been claimed to be acquired originally because of neovascularization secondary to chronic infection. This case is congenital in origin because of the abnormal artery originating from the circumflex artery.

In addition, ELS is frequently localized in the left lower lobe, just between the lobe and the diaphragm in its own pleural envelope. The type of pleural envelope in the lesion (IL-EL) cannot be detected by CT scanning alone but requires histopathologic verification. The venous drainage in this case also has not been clearly identified. Pulmonary venous drainage is characteristic in patients with ILS. When the diagnosis is uncertain, surgery is the rule in sequestrations.13,14 Two other limitations of this case are the discordance with the acquired ILS hypothesis of Stocker and Makzak,2 in that arteries do not reach the lesion via the ligamentum pulmonale, and the lack of a bronchologic study. There are ILS cases with communication to the tracheobronchial tree or cases in which inflammatory changes occur in adjacent bronchi.19 In this case, the patient has a history of bronchitis of quite a long duration. Collateral arteries from the aortic arch to the lesion are not proof enough that the lesion was acquired. Besides, by means of collateral ventilation, the sequestrated portion of the lung in patients with ILS can be ventilated and infected.20 The above-mentioned sequential events do not necessarily mean that ILS is acquired as a result of infection.

I believe that this case of sequestration is congenital in origin, and, with respect to the above-mentioned reasons, it will not be
objective to define the etiology of this case as both congenital and acquired unless further investigations concerning venous drainage, type of pleural envelope of the lesion, and aortic vessels to the lesion, as well as bronchologic studies, are performed.

Attila Saygı, MD
Heybeliada Chest Diseases and Thoracic Surgery Center 
Istanbul, Turkey

Correspondence to: Attila Saygı, MD, Atatürk Caddesi Sehit Ilhan, Sokak Demireci Apt No. 8/4, Sakaraycilar, Kadıkoy, Istanbul 81300, Turkey; e-mail: oguzsaagi@superonline.com

REFERENCES

3 Kaemmerlen JT. Sequestrated information. Chest 1984; 86:611–615
16 Lane SD, Burkott H, Scott HW. Congenital BPFM. Radiology 1971; 101:291–292

To the Editor:

We appreciate Dr. Attila Saygı’s interest and comments regarding our Roentgenogram of the Month in June 2000 demonstrating sequestration.1 Briefly, our patient was a 66-year-old Chinese man with a long history of recurrent bronchitis/chest infections who resided at high altitude and who traveled to Washington, DC for evaluation of his dyspnea. We discovered a left lower lobe infiltrate by plain chest radiography, a large aberrant artery arising from the left circumflex artery leading to the left lower lobe, and multiple collateral vessels arising from the aortic arch also traversing to the left lower lobe. We believe that this case represents an intralobar sequestration of both congenital and acquired etiology.

Although it is somewhat difficult to fully ascertain Dr. Saygı’s arguments, it appears that he disagrees with our conclusion that there was both a congenital and acquired origin of the abnormal vasculature. He proposes that the aberrant circumflex artery as well as the multiple collaterals from the aortic arch are congenital in origin, primarily citing the theory of Clements and Warner2,3 regarding congenital acquisition of intralobar pulmonary sequestration.

As stated in our earlier discussion, there is undoubtedly a debate in the literature regarding the etiology of intralobar sequestrations as congenital or acquired anomalies. There are compelling theories that argue in favor of the acquired origin of intralobar sequestration4,5 and also those in favor of the congenital theory.2,3 Stocker and colleagues4,5 argue that there are small systemic arteries in the pulmonary ligament that can be parasitized to supply an infected region of the lung when the pulmonary artery supply is compromised. Livingston et al6 also argue that chronically infected lung tissue can lead to neovascularization from high-pressure systemic circulation. We concede that in our patient the region of the abnormal lung was already being supplied by a high-pressure systemic artery originating from the circumflex artery, rather than from the low-pressure pulmonary artery as in normal anatomy. Also, in our patient there was no evidence of compromise of the aberrant circumflex vessel. We believe that the chronic infection, inflammation, and fibrosis that originated from the underlying sequestrated lung tissue and then from recurrent infection led to neovascularization in the form of multiple collaterals arising from the aortic arch. We argue that arterial vasculature from congenital and acquired origins can coexist in a sequestration within the same patient.

This patient’s symptoms spontaneously improved after his arrival in Washington, DC, and he quickly returned to his homeland. Therefore, we were unable to perform further diagnostic studies or therapeutic interventions, such as determining the venous drainage, or performing bronchoscopy or bronchography, or thoracic surgery to accurately define the pleural envelope. Had we been able to perform some of these additional studies, in the very least, we would have obtained more data regarding this unusual case of sequestration.

We agree that the pathogenic theory of intralobar sequestration remains a controversial issue.

Mary Margaret Lewis, MD
Edward Tsou, MD, FCCP
Georgetown University Medical Center
Washington, DC

Correspondence to: Mary Margaret Lewis, MD, Pulmonary and Critical Care Medicine, Georgetown University Medical Center, 3800 Reservoir Rd NW, Washington, DC 20007

REFERENCES

2 Clements BS, Warner J. Pulmonary sequestration and related congenital bronchopulmonary-vascular malformations: nomenclature and classification based on anatomical and em-