abscess. The abscess was drained and the gallstones were extracted percutaneously under fluoroscopic guidance.

We describe the first case of a sterile empyema that resulted from an abscess surrounding spilled gallstones which eroded into the diaphragm 4 months following a laparoscopic cholecystectomy. Furthermore, we were able to definitively diagnose and treat the complex effusion and remove the gallstones using a single, minimally invasive surgical procedure. Interestingly, in this and both previously described reports, the pleuropulmonary complication occurred 3 to 4 months following the laparoscopic cholecystectomy.8,9 Given the frequency of retained gallstones, we propose they be included in the differential diagnosis of a pleuropulmonary process that occurs in the ensuing months following a laparoscopic cholecystectomy.

In summary, this report suggests that spilled gallstones from laparoscopic cholecystectomy may lead to pleuropulmonary complications several months following the surgery. Thoracoscopy may be a useful procedure for the treatment of pleural, diaphragmatic, and subdiaphragmatic processes related to retained gallstones.

**REFERENCES**


**Anomalous Systemic Arterial Supply to Normal Basal Segments of the Left Lower Lobe**

**A Report of Two Cases**

Takashi Hirai, MD; Yolsuke Ohtake, MD; Shin Mutoh, MD; Masato Noguchi, MD; and Akira Tamanaka, MD

Two cases of anomalous systemic arterial supply to the basal segments of the left lower lobe without sequestration are presented. In the first case, the final diagnosis was made during a surgical operation, and lobectomy of the lower lobe of the left lung was performed. In the second case, the preoperative diagnosis made by CT was confirmed by angiography. An anastomosis was performed between the anomalous artery and the pulmonary artery without resection of the basal segments. Six months after surgery, pulmonary angiography showed improved flow of the anastomosed vessel, but little improvement was evidenced in the perfusion scan. (CHEST 1996; 109:286-89)

**Key words:** anomalous artery; sequestration; systemic arterial supply to the lung; vascular anastomosis

Anomalous systemic arterial supply to the basal segments of the lower lobe of the left lung without sequestration is a rare congenital abnormality with characteristic roentgenologic features. We treated two patients with this lung anomaly. In one, the final diagnosis was made during a surgical operation, and lobectomy of the lower lobe of the left lung was performed. The second patient’s initial diagnosis was aided by CT, and pulmonary arterial and aortic angiography confirmed the diagnosis. The anomalous artery was Anastomosed to the pulmonary artery of the upper lobe of the left lung without resection of the basal segments.

**REPORT OF CASES**

**Case 1**

A 29-year-old man was referred to our hospital because of hemoptysis. Radiographs of the chest and CT showed a retrocardiac nodular shadow. Transbronchial biopsy was attempted via bronchoscopy, but the biopsy forceps could not reach the lesion. Tuberculosis was suspected, and antituberculous agents were administered for 3 months. However, the size of the nodule was unchanged. Exploratory thoracotomy was performed to rule out lung cancer. A large systemic artery (maximum diameter, 3 cm) from the descending thoracic aorta supplied the basal segments of the lower lobe of the left lung. The aberrant artery was ligated, and a lobectomy of the lower lobe of the left lung was performed. The postoperative course was uneventful. Histologic examination showed an elastic-walled artery.

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Figure 1. Chest CT scan (case 2). A nodular shadow (3.5 x 3.0 cm) is seen beside the descending thoracic aorta.

Case 2

A 30-year-old asymptomatic man was referred to our hospital for evaluation of an abnormal shadow evidenced on a chest radiograph. His family history and past medical history disclosed no pertinent findings. On physical examination he appeared to be healthy, without cyanosis, clubbing, or cardiac murmur. With the patient breathing room air, the PaO₂ value was 95 mm Hg. The chest radiograph revealed a retrocardiac nodular shadow in the lower field of the left lung. A CT of the chest showed a nodular shadow beside the descending thoracic aorta (Fig 1). A technetium-99m (99mTc) lung perfusion scan revealed a perfusion defect in the left basal segments. Aortography showed an anomalous artery from the descending thoracic aorta entering the basal segments of the lower lobe of the left lung which drained into the inferior pulmonary vein (Fig 2, top). Pulmonary arteriography showed a complete lack of pulmonary arterial supply to these same segments (Fig 2, bottom). Bronchoscopy findings were normal. A diagnosis of anomalous systemic arterial supply to the basal segments without sequestration was made. At operation, the visceral pleura of the lower lobe of the left lung was covered with many spider telangiectases. An anomalous vessel arose from the descending thoracic aorta. At its origin, the vessel was 8 mm in diameter. The left pulmonary artery appeared to supply only the upper lobe of the left lung. The anomalous vessel was transected at its origin, and the proximal end was oversewn with 3-0 Prolene. End-to-side anastomosis between the anomalous artery and the pulmonary artery was performed with 5-0 Prolene without resection of the basal segments. A small section of the lung (S8) was removed for histologic examination.

The anomalous artery was elastic-walled, and the lung parenchyma was fibrotic. There were scattered thrombi in the peripheral pulmonary artery (Fig 3). Anticoagulation treatment with warfarin sodium and antiplatelet therapy with low-dose aspirin were begun.

Figure 2. Top: aortography (case 2). Aortography shows an anomalous systemic artery arising from the descending thoracic aorta. Bottom: pulmonary arteriography (case 2). No pulmonary artery to the left basal segments is seen.
after surgery. Four weeks after surgery, a $^{99m}$Tc lung perfusion scan and pulmonary arteriography revealed no improvement of circulation to the lower lobe of the left lung. Anticoagulation and antiplatelet therapy were therefore continued. After 6 months, subsequent pulmonary angiography showed that the flow into the lower lobe of the left lung improved. However, a lung perfusion scan revealed little improvement in that value. Anticoagulation was continued for 6 more months because of delayed imaging of the anastomosed vessel. One year after surgery, the patient had no respiratory symptoms. With the patient breathing room air, the PaO$_2$ level was 103 mm Hg.

**DISCUSSION**

This disease was previously classified as Pryce type I sequestration, an abnormal artery (systemic) to normal lung. However, it differs considerably from the definition of sequestration because it has normal bronchial connections. Sade et al. introduced the concept of a "sequestration spectrum" to include various combinations of abnormal bronchus, arterial supply, and venous drainage. At one end of the spectrum is an anomalous vascular supply to a nonsequestrated lung. At the other end of the spectrum is abnormal pulmonary tissue similar to that found in sequestration but without anomalous vascular supply, namely, bronchopulmonary cyst. Between these two extremes lie the variants of sequestration. Clements and Warner classified this clinical entity as "arterial pulmonary malinosculcation," namely an aberrant systemic supply to an area of otherwise normal lung.

In our first patient, a mistaken diagnosis of pulmonary tuberculosis initially was made. Additional imaging studies such as CT or MRI may have allowed a preoperative diagnosis. In our second patient, the preoperative diagnosis was made by CT and was confirmed by aortography and lung perfusion scan. If this anomaly is kept in mind, the diagnosis is not so difficult because the findings of CT are characteristic: a retrocardiac nodular (vascular) shadow connected to the descending aorta entering the basal segments of the lower lobe of the left lung. Enhanced helical CT was very useful for the initial diagnosis. A surgical operation is indicated for all patients with this anomaly because it has potential risks, such as hemothysis due to pulmonary hypertension, heart failure due to left-to-left shunt, and infection. In most reported cases of this disease, lobectomy or segmentectomy was performed. We performed a lobectomy of the lower lobe in our first patient, and in the second we anastomosed the anomalous artery to the pulmonary artery of the upper lobe of the left lung without resecting the basal segments. Hessel et al. first reported the successful transfer of the origin of the anomalous vessel to the pulmonary artery in a 5-year-old boy and described pulmonary angiography which revealed normal flow into the lower lobe of the left lung 4 months after surgery. To our knowledge, our case is the second one to be reported in the English-language medical literature of such a transfer of a lobar artery of systemic origin. In most reported cases histologic examination has shown an anomalous elastic-walled vessel and the lung parenchyma with fibrosis due to pulmonary hypertension. In our second case, circulation to the basal segments was not improved initially, presumably because of such histologic changes. Anticoagulant therapy was necessary for 1 year until flow through the vessel improved. Six months after surgery, flow in the anastomosed vessel improved, but the perfusion scan improved only slightly. A prolonged follow-up period is necessary to determine the value of this procedure.

In summary, we have described two cases of anomalous systemic arterial supply to the basal segments of the lower lobe of the left lung without sequestration. An accurate diagnosis can be made with contrast CT and can be confirmed by angiography. Long-term follow-up is necessary to determine the efficacy of vascular anastomosis.

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Subacute Tricuspid Regurgitation With Severe Hypoxemia Complicating Blunt Chest Trauma*

Flavio Ribichini, MD; Riccardo Conte, MD; Antonio Lioi, MD; Antonio Dellavalle, MD; and Giovanni Ugliengo, MD

A case is reported on subacute tricuspid regurgitation due to rupture of papillary muscles, with right-to-left shunting, in the absence of extracardiac lesions after blunt chest trauma.  (CHEST 1996; 109:289-91)

TR=tricuspid regurgitation; R-L=right-to-left; PFO=patent foramen ovale

Key words: subacute papillary muscle rupture; traumatic heart injury; tricuspid regurgitation

Cardiac injury after blunt chest trauma was well studied in the 1950s, and the diagnosis and treatment of rupture of the ventricular walls and interventricular septum have been described. Severe hypoxemia is observed commonly after blunt chest trauma and usually is the result of lung damage and intrapulmonary shunting.2 Traumatic tricuspid regurgitation (TR) with intracardiac right-to-left (R-L) shunting also has been reported as a rare finding3,4; in all cases TR was accompanied by severe bony or visceral lesions, and R-L shunting was explained by elevated right atrial pressures in the presence of a patent foramen ovale (PFO).

A case of TR with R-L shunt in the absence of extracardiac lesions and with no elevation of mean right atrial pressure is described.

CASE REPORT

A 43-year-old man was admitted to the hospital after a severe vehicle accident in which he was squeezed against the steering wheel. He complained of low-grade pain in the anterior area of the chest. He was in no distress, his arterial blood pressure was 110/60 mm Hg, with a pulse of 85 beats per minute and a respiratory rate of 20 breaths per minute. A grade 1/6 systolic murmur was audible at the apex. The rest of the physical examination disclosed no abnormalities. A chest X-ray film was normal. The ECG showed rotation of the electrical axis to the right, with poor R-wave progression in the precordial leads and ST-T segment changes of uncertain significance. No previous ECG was available for comparison. Two days later, neither physical examination findings nor the ECG showed changes. The patient felt very well and decided to leave the hospital against medical advice. He was admitted 10 days later due to increasingly severe shortness of breath and fatigue after a week of well-being. Examination revealed tachypnea, acrocyanosis, and a grade 2-3 systolic murmur at the apex. The ECG was unchanged, as was the chest X-ray film. Blood chemistry values were normal. Arterial blood gas value analysis revealed hypocapnic hypoxemia (PaO₂, 48 mm Hg; PaCO₂, 16 mm Hg) with the patient breathing

<table>
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<th>Part of Heart</th>
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*Abbreviations: Ao=aorta; SVC=inferior vena cava; LV=left ventricle; PA=pulmonary artery; PCW=pulmonary wedge pressure; RA=right atrium; RV=right ventricle; SVC=superior vena cava; Qp= pulmonary flow; Qs=systemic flow.

Figure 1. Right ventriculogram in the posteroanterior projection. Prompt opacification of the left atrium is observed. LA=left atrium; PA=pulmonary artery; RA=right atrium; RV=right ventricle.