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Surgical Management of Infants with Congenital Lobar Emphysema and Concomitant Congenital Heart Disease

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ABSTRACT

Objective: Congenital lobar emphysema (CLE) is an uncommon cause of infantile respiratory distress. It is diagnosed on the basis of evidence of lobar overaeration, mediastinal shift, and compression of the adjacent lobe. Concomitant congenital heart disease (CHD) and CLE is not uncommon. In the literature a 12% to 20% concomitance rate is given. The optimal treatment of respiratory symptoms associated with CLE and CHD is not clear; however, there has been a great deal of progress in the treatment of CLE and CHD. The aim of this study was to evaluate a clinical experience with and long-term follow-up of the surgical treatment of 13 patients with concomitant CLE and CHD.

Material and Methods: We reviewed the cases of 13 patients with concomitant CLE and CHD. The medical records were evaluated with reference to age, type of CHD, pulmonary artery pressure, clinical symptoms, and results of surgical management.

Results: One patient died. This patient had ventricular septal defect (VSD) and left upper lobe emphysema in the postoperative period. The remaining patients undergoing follow-up were clinically well at the final evaluation. Postoperative thoracic computed tomography revealed complete spontaneous regression of emphysema 3 months after division of ductus arteriosus in 1 patient. Pulmonary hypertensive episode was seen in 3 patients after the early postoperative period. Five of the patients were discharged with bronchodilator treatment after surgery. Six patients needed positive inotropic support. Among the patients with pulmonary hypertension and those with VSD who had undergone cardiopulmonary bypass, we found a greater need for inotropic support, a higher risk of postoperative infection, and a longer intubation period. Echocardiography in the late postoperative revealed decreased pulmonary artery diameter and pressure; myocardial performance was normal. Results of blood gas analyses revealed increased oxygen saturation and decreased partial pressure of carbon dioxide. Normal exercise activity was found in all patients.

Discussion: The presence of CHD, especially in infants with unusual respiratory distress symptoms, should be kept in mind, and echocardiography and/or cardiac catheterization should be considered in the diagnosis. In patients with high pulmonary artery pressure, palliative or corrective surgery for CHD in addition to lobectomy can be considered. We believe that for lesions without high pulmonary artery pressure, such as small atrial septal defect and patent foramen ovale, clinical follow-up is sufficient treatment after lobectomy. If the cause of CLE is compression of large ductus arteriosus, only division of the patent ductus arteriosus may be considered before lobectomy and clinical and radiologic follow-up. The cardiac lesion should be assessed as to severity and ease of management. A corrective procedure can be carried out at lobectomy. Because of the technical ease with which the cardiac operation can be performed at the time of lobectomy, we suggest that in addition to lobectomy, operative treatment of cardiac lesions be performed.

INTRODUCTION

Congenital lobar emphysema (CLE) is a rare clinical condition that often necessitates lobectomy in early infancy. The most frequent site for emphysema is the right middle lobe, where the right middle bronchus is often compressed by dilated pulmonary vessels. Some of our patients had no dilated pulmonary vessels, but 2 had dilated right ductus arteriosus, which is near the left main bronchus. Bronchial cartilaginous dysplasia and check valve mechanisms are often associated with this condition, but in one half of affected infants, the etiology remains undetermined. Extrinsic obstruction, such as dilated pulmonary arteries associated with ventricular septal defect (VSD), patent ductus arteriosus (PDA), or tetralogy of Fallot with dilated pulmonary arteries can cause this pathologic condition. We describe 13 infants with concomitant congenital heart disease (CHD) and CLE. In 12% to 20% of patients CHD is associated with lobar emphysema and usually results from the association of hypertensive or dilated pulmonary arteries with VSD or PDA. However, we found no information about the long-term follow-up of these patients. In our patients, emphysema was found in the right upper or left lobe in cases of lobar

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Table 1. Clinical and Radiographic Results

Number of patients	13
Age range at operation	56 d–24 mo
Female/male ratio	8/5
Affected side	
Left upper lobe	8
Right upper lobe	5
Chest x-ray	
Hyperlucency	10
Shift to opposite side	4
Atelectasis	5
Pneumothorax	—
Thoracic computed tomography	
Emphysema	11
Shift/Herniation to opposite side	5
Compression of the adjacent lobe	5
Arterial blood gas analysis*	
Hypoxia (≤60 mm Hg)	9
Hypercapnia (≥50 mm Hg)	8

*Room air.

emphysema with CHD, and it was present at long-term follow-up evaluation.

PATIENTS AND METHODS

Thirteen patients with the diagnosis of concomitant CLE and CHD were followed up in the Hacettepe University Department of Thoracic and Cardiovascular Surgery from 1987 to 2003. The ages of the 8 girls and 5 boys varied from 26 days to 19 months. The mean age was 8.1 months. The mean age at operation was 9.5 months (range, 56 days to 24 months). The minimum, maximum, and mean follow-up durations of all patients were 83.3 months, 22.6 months, and 46.3 ± 25.6 months, respectively. Echocardiography, cardiac catheterization, and computed tomography (CT) of the thorax were performed in all patients. On chest x-ray films, emphysema was defined in 11 patients, and contralateral shift of the mediastinum and lung atelectasis were found in 4 patients. Clinical and radiographic results are summarized in Table 1.

Three patients had patent foramen ovale (PFO) with normal pulmonary artery pressure, 1 patient had cyanotic CHD (pulmonary atresia and VSD), and 7 patients had severe or moderate pulmonary hypertension (PH). Lobectomy, pulmonary artery banding, and left modified Blalock-Taussig shunt with a 4-mm thin-walled Gore-Tex polytetrafluoroethylene vascular graft were performed in 1 patient. Ligation of PDA and left upper lobectomy were performed in 2 patients. Compressive left upper lobe emphysema was diagnosed because of the presence of a large PDA at thoracic CT. Division of PDA was performed in this patient with PDA and left upper lobe emphysema. Spontaneous regression of left upper lobe emphysema 6 months after surgery was found in this patient (Figures 1 and 2). Total correction was performed in a patient with PDA and aortopulmonary window (APW) and one with VSD (Figures 3 and 4).

The left upper lobe was affected in 8 (71.6%) of the patients and the right upper lobe in 5 (29.4%) of the patients. Clinical and laboratory findings (including mean pulmonary artery pressure) at the time of diagnosis and surgical results are summarized in Table 2.

The diagnosis of concomitant CLE and CHD with or without PH was based on the findings of clinical invasive or noninvasive radiological studies, clinical examination, and arterial blood gas analysis along with historical confirmation in patients who underwent surgery at the time of diagnosis. Types of cardiac defects concomitant with CLE are shown in Table 2.

Hyperlucency was observed in all patients except 1 who had VSD and PH. Cardiomegaly and contralateral shift of the lung were other radiological findings. CT of the thorax was performed in all cases and revealed emphysema of the affected lobe and shift to the opposite side. Arterial blood gas analysis showed hypercapnia and hypoxia, especially in patients who had moderate or severe PH. Results of thoracic CT and cardiac catheterization were recorded. Patients with

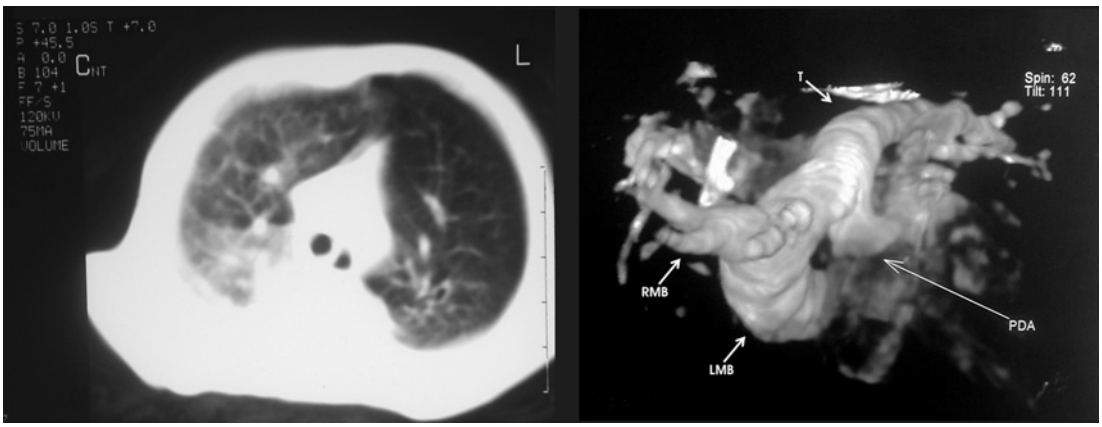

Figure 1. Left, Chest computed tomographic (CT) scan shows left upper lobe emphysema. Right, 3-dimensional thoracic CT scan shows compression of the left main bronchus by a large ductus arteriosus (originating from descending aorta). Long arrowhead indicates left main bronchus compressed by large patent ductus arteriosus (PDA); LMB, left main bronchus; RMB, right main bronchus; T, trachea.

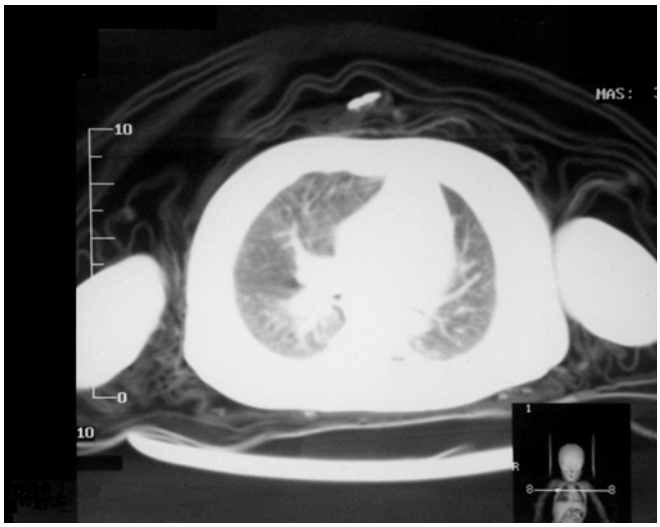

Figure 2. Postoperative thoracic computed tomographic scan shows spontaneous regression of lobar emphysema 3 months after surgery.

severe respiratory distress, hypoxia, and moderate or severe PH were surgically treated. Single lobectomy was performed in 4 patients, 3 of whom had PFO and 1 of whom had secundum type atrial septal defect (ASD) with pulmonary sequestration. Extrapulmonary sequestration of the right lung with emphysema of the right upper lobe was found in this patient (Figure 5, left). Pulmonary sequestration originated in the celiac artery. Because of the high mortality rate for excision of pulmonary sequestration by laparotomy, coil embolization was performed and soon increased the blood supply to the sequestered part of the lung (Figure 5, right). Decortication was performed later, but lobectomy was not performed because compression of the adjacent lobe was not defined. The right lung expanded in the postoperative period, and chest x-ray findings were normal. Mediastinal shift was reduced compared with the findings at preoperative CT of the thorax. Lobectomy and correction of the cardiac defect were performed through right thoracotomy in the other 7 patients. Cardiopulmonary bypass (CPB) was used for only 1 patient who had VSD. After closure of VSD the patient could not be weaned from CPB, so left upper lobectomy was performed because the patient had persistent respiratory acidosis. Persistent lobar emphysema was relieved in the postoperative period, and the patient was successfully weaned from the ventilator.

We preferred thoracotomy for most of the patients. The exceptions were those who had VSD and APW-PDA together. Midline sternotomy was performed in these patients. Fentanyl was used for postoperative sedation, especially for the patients who had PH. Transcutaneous oxygen saturation was measured with a pulse oximeter in the preoperative period and continuously in the postoperative period. Respiratory acidosis, bronchospasm, low cardiac output, respiratory system infection, and atelectasis due to prolonged intubation were the most common postoperative problems. Postoperative problems and treatment strategies are summarized in Table 3.

RESULTS

Lobectomy was performed on 1 patient with concomitant CLE and VSD. We planned correction of VSD within 2 months, but the patient died at home of undetermined causes during the follow-up period after lobectomy.

Only 1 patient (patient 5) died. This patient had VSD and left upper lobe emphysema in the postoperative period. The other patients undergoing follow-up were clinically well at the final evaluation. The left upper lobe was most often affected (68.3% of cases) followed by the right upper lobe (31.7% of cases). The female/male ratio (1/2) was similar to that reported in the literature. PH episodes in the postoperative period occurred in patients 5, 9, and 10. Sedation was obtained with anesthetic drugs such as midazolam (Dormicum)

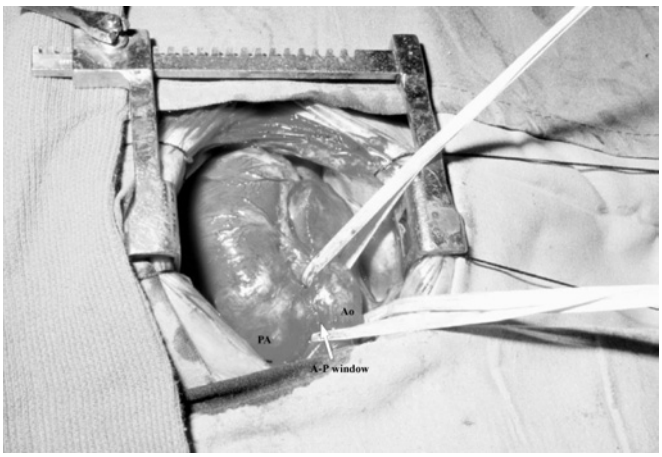

Figure 3. View of aortopulmonary window through midline sternotomy before repair. PA indicates pulmonary artery; Ao, aorta; A-P, aortopulmonary.

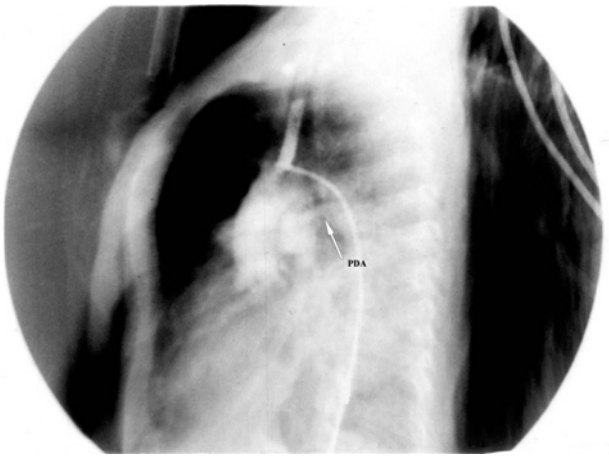

Figure 4. View from left heart catheterization shows patent ductus arteriosus (PDA) and large distance from anterior to posterior chest wall due to emphysematous upper lobe.

Table 2. Cardiac Defects Concomitant with Congential Lobar Emphysema and Surgical Results*

Patient	Cardiac Defect	Affected Lobe	Age at Operation	Operation	Surgical Result	Mean PAP, mm Hg
1	PFO, CPD	LUL	11 mo	Lobectomy	Good	8
2	PFO	LUL	56 d	Lobectomy	Good	12
3	PFO	LUL	2 mo	Lobectomy	Good	11
4	PDA, APW, PH	LUL	18 mo	Lobectomy, closure of APW, ligation of PDA without CPB	Good	45
5	VSD, PH	LUL	5 mo	Closure of VSD, lobectomy	Died	48
6	ASD, PDA, PH, extrapulmonary sequestration	RUL	6 mo	Decortication and coil embolization	Good	20
7	VSD, Pulmonary Atresia	RUL	15 mo	Right modified Blalock-Taussig shunt, lobectomy	Good	—
8	Small VSD, PH	RUL	7 mo	Lobectomy	Good	18
9	VSD, PDA, PH	RUL	8 mo	Ligation of PDA, lobectomy	Good	44
10	VSD, PH	LUL	7 mo	Pulmonary artery banding, lobectomy	Good	52
11	Small VSD, PDA, PH	LUL	9 mo	Ligation of PDA, lobectomy	Good	28
12	Secundum ASD	RUL	2 y	Lobectomy	Good	17
13	PDA	LUL	9 mo	Division of PDA	Good	22

*PAP indicates pulmonary artery pressure; PFO, patent foramen ovale; CPD, congenital pericardial defect; LUL, left upper lobe; PDA, patent ductus arteriosus; APW, aortopulmonary window; PH, pulmonary hypertension; CPB, cardiopulmonary bypass; VSD, ventricular septal defect; ASD, atrial septal defect; RUL, right upper lobe.

and fentanyl. Prostaglandin I_2 (PGI_2) (Ilomedin; Schering, Berlin, Germany), 20 μg/mL, was administered to this group because they had severe PH and to prevent PH episodes. We attempted to maintain pH at 7.35 to 7.45, O_2 saturation between 90% and 95%, and P_{CO_2} at 35 to 40 mm Hg. Upper respiratory airway infection developed on the fifth postoperative day in patient 4. Because of infection and laryngeal edema, he was extubated 11 days after surgery. In this patient and the other 11 patients, PGI_2 treatment was given in nasal spray form before and after extubation. Echocardiographic results showed ventricular function was normal and pulmonary infection was relieved. The patient was discharged with bronchodilator treatment 15 days after surgery. In patients 4, 5, 6, 7, 9, and 10, inotropic support was given in the early postoperative period. In 3 cases inotropic support was stopped early, whereas in 4 patients the treatment was given for 4 more days. In the patient group that was given inotropic support, we observed that postoperative extubation was later and stay in the intensive care unit was longer than for the remaining patients (intubation time, 9 ± 3 days; average, 4.8 days). Especially for patients with PH and a patient with VSD for whom CPB was used (patients 4, 5, 9, 10, and 11), we observed that need for inotropic support and postoperative infection risk were greater and that the intubation period was longer than for the other patients.

Chest x-rays were either normal or showed hyperlucency due to compensatory hyperaeration on the side operated on. At the late postoperative evaluation, echocardiography revealed decreased pulmonary artery diameter and pulmonary artery pressure and normal myocardial performance.

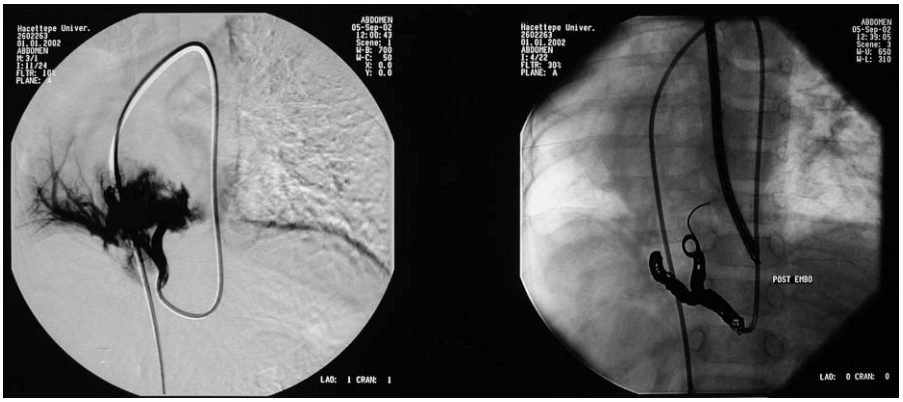

Figure 5. Left, Extrapulmonary sequestration of the right lung with emphysema of the right upper lobe in a patient with secundum type atrial septal defect with pulmonary sequestration. Right, Successful coil embolization in the same patient. Pulmonary sequestration originated in the celiac artery. Coil embolization was preferred because of the high mortality rate for excision of pulmonary sequestration by laparotomy.

Table 3. Postoperative Problems and Treatment Strategies*

Postoperative Problem	Patient	Treatment Strategy
Low cardiac output	5, 6, 7, 9, 10	Positive inotropic support with dopamine and dobutamine
Pulmonary hypertensive crisis	7, 10	Sedation with fentanyl; prostaglandin I_2 (Ilomedin) administration 0.5 ng/kg per hour
Postoperative bronchospasm	4, 6, 7, 10, 12	Bronchodilator (Salbutamol) and prostaglandin I_2 nasal spray administration.
Recurrent or persistent atelectasis	6, 7, 10, 11, 12	Endobronchial lavage with bronchoscopic guidance in patients 4, 5 and 11; postural drainage in the others
Airway infection	10, 11	Prolonged antibiotic administration, bronchodilator treatment
Respiratory or metabolic acidosis	4, 5, 7, 10, 11	Nasal CPAP, bicarbonate administration
Prolonged intubation time	4, 5, 9, 10, 11	Prostacyclin and bronchodilator administration; sedation

*CPAP indicates continuous positive airway pressure.

Results of blood gas analyses revealed increased oxygen saturation and decreased partial carbon dioxide pressure. Normal exercise activity was observed in all patients.

DISCUSSION

CLE is an unusual cause of respiratory distress diagnosed on the basis of overaeration and compression of the adjacent lobe. Traditional treatment of CLE is lobectomy of the affected lobe, which is associated with collapse of the adjacent lung or mediastinal shift with respiratory distress [Kennedy 1965, Murray 1967, Lincoln 1971]. In 1945 Gross and Lewis published the first case report of CLE treated by lobectomy [Gross 1945]. Some authors reported that in a few studies patients were treated by conservative medical means [Roghair 1972, Man 1983, Stigers 1992, Ozcelik 2003]; however, Dogan et al suggested lobectomy for patients with severe onset and symptoms that were resistant to medical treatment [Dogan 1997]. CLE is more difficult in that the differential diagnosis includes pneumatocele, pneumothorax, lung hypoplasia, atelectasis with hyperinflation of the contralateral lung, and cystic adenomatoid malformation.

The etiologic mechanism proposed for CLE is based on the unusual flaccidity of the bronchial wall caused by the abnormal or malformed bronchial cartilage, redundant bronchial mucosal folds and webs, or compression of the bronchus by an abnormal vessel [Stranger 1969, Pierce 1970]. According to some investigators, CLE is caused by the check-valve mechanism created by compression of the bronchus by the pulmonary artery distended as the result of PH [Isojima 1978]. A mechanism by which CHD may cause CLE was suggested by Stranger et al [1969], who found that enlarged pulmonary arteries or an enlarged left atrium can compress the lobar bronchus. In association with this external compression, poorly developed cartilaginous rings of the infantile bronchus form. Our male/female ratio was similar to that reported in the literature. CLE usually affects the left upper lobe, followed by the right middle lobe. In our study, however, the left upper lobe was affected most often, followed by the right upper lobe (68.3% and 31.7% of cases, respectively).

CHD is found in 12% to 20% of patients with CLE [Roguin 1980]. Murray [1971] reported a 14% incidence of associated CHD and lack of familial incidence. Concomitant CLE and CHD is found in approximately 15% of patients in our clinic, a finding identical to that in the literature. Associated anomalies are pulmonary and cardiovascular, such as PDA, VSD, and tetralogy of Fallot with pulmonary atresia, which causes a predisposition to compression of the tracheobronchial tree by dilated pulmonary arteries [Pierce 1970, Litwin 1973]. CLE was frequently concomitant with CHD and PH in our patients. VSD has been most commonly associated with CLE in cases of emphysematous right middle lobe [Stranger 1969]. In our cases, VSD and PFO most frequently accompanied CHD.

Respiratory distress in patients with cyanotic CHD occurred in the first year of life, especially between 2 and 9 months of age. Symptoms of concomitant CHD and CLE started at an earlier age than CLE alone.

The optimal treatment of respiratory distress associated with an emphysematous lobe, collapsed adjacent lung, and CHD is not clear [Rudolph 1969]. Surgical alternatives include correcting the cardiac defect alone, resection of the affected lobe, and correction of both lesions [Pierce 1970, Isojima 1978, Jones 1965, Gordon 1990].

The results of the surgical treatment of both pathologic conditions in the same operation have not yet been predicted. We need to evaluate a larger patient population. Our experience, however, showed that in infants who had concomitant acyanotic CHD and CLE, the emphysematous lobe caused pressure on the other lobe, the normal lobe collapsed, and the collapsed lung caused respiratory insufficiency. In the third case, an infant with CLE and ASD had

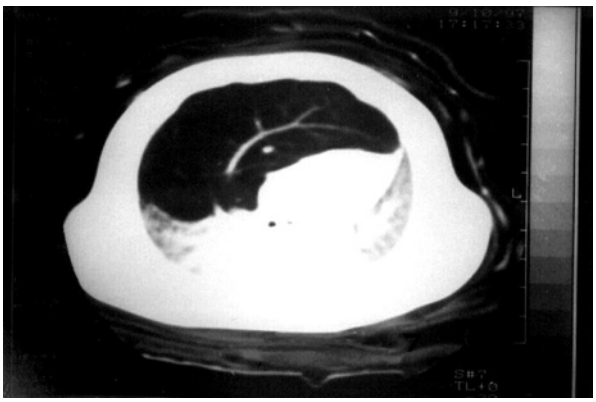

Figure 6. Preoperative computed tomographic image shows right lower lobe emphysema.

right lower lobe sequestration (Figure 6). The right lung was totally collapsed, and the emphysematous lobe at risk because of the collapse had to be returned to normal function. We perform coil embolization on this kind of patient. This method saves the patient from the risk of mortality and morbidity due to laparotomy. The respiratory distress originates from the collapsed lung.

Pierce and his colleagues reported that in their series in which the patients had both a congenital heart defect and CLE, the lung was not collapsed. Those authors corrected only the pathology. In addition, Elmacı and his colleagues [2001] described a patient with both VSD and CLE. In that case, 4 years after closure of the VSD, it was found that the emphysematous lung area was normal. On the basis of these 3 cases, we believe that congenital heart defects and CLE can be corrected together. The patients easily tolerate extubation, and respiratory function returns to normal with early mobilization. Our patients, especially those with severe PH, were sedated for at least 24 to 48 hours before extubation, pulmonary wedge pressure levels were monitored continuously, and intravenous prostacyclin infusion was given to patients with high pulmonary pressure levels. At the same time, congenital heart defects with right to left shunts can cause postoperative infective endocarditis, and high pulmonary artery pressure can have adverse effects on respiratory function that soon affect the growth pattern. Especially in infancy, high pulmonary artery pressure leads to hyperaeration, which can be diagnosed with a chest x-ray. In those cases, CLE should be considered, and CT of the thorax should be performed.

We believe that in patients with CLE, especially with high pulmonary artery pressure, lobectomy should be considered to control respiratory distress and prevent PH. It is clear that with those results, palliative cardiovascular surgery should be performed on infants with atrioventricular canal defect, PDA, and VSD with PH and/or decreased pulmonary blood flow in cyanotic heart disease. Palliative operations such as pulmonary artery banding, Blalock-Taussig shunt, and ligation of PDA and APW have relieved the irreversible pulmonary and alveolar damage caused by the disease and have contributed to infant oxygenation after surgery. The presence of CHD also should be kept in mind for infants with unusual severe pulmonary symptoms, and echocardiography and/or cardiac catheterization should be considered in the diagnosis because 12% to 20% concordance is present. In 7 patients with high pulmonary artery pressure, we performed palliative or corrective surgery in addition to lobectomy. We believe that for patients without high pulmonary artery pressure levels, such as those with small ASD, PFO, or small ductus, clinical follow-up is sufficient treatment after lobectomy. PFO and ASD were found to have closed in the long-term follow-up of our patients with PFO and ASD.

In summary, the cardiac lesion should be assessed as to severity and ease of management, and a corrective procedure can be carried out at the time of lobectomy. We propose that surgeons and clinicians keep in mind the presence of CLE if there are unexpected findings in infants with CHD, such as severe cyanosis and diminished exercise tolerance. Although abnormal heart sounds, tachycardia, hypoxia, and cyanosis are the main symptoms in infants with CHD, these symptoms can be severe and appear early in infancy. We suggest that because of the technical ease with which the cardiac operation can be performed, cardiac lesions be treated at the time of lobectomy.

REFERENCES

Dogan R, Demircin M, Sarigul A, Pasaoglu I, Gocmen A, Bozer AY. 1997. Surgical treatment of congenital lobar emphysema. Turkish J Pediatr 39:35-44.

Elmacı TT, Guler N, Aydogan U, Onursal E. 2001. Infantile lobar emphysema and tracheal bronchus in a patient with congenitally heart disease. J Pediatr Surg 36:1596-8.

Gordon I, Dempsey JE. 1990. Infantile lobar emphysema in association with congenital heart disease. Clin Radiol 4:48-52.

Gross RE, Lewis JE Jr. 1945. Defect of the anterior mediastinum successful repair. Surg Gynecol Obstet 80:549-54.

Isojima A, Yuasa H, Kusagawa M, et al. 1978. Surgical treatment of infantile lobar emphysema in cardiovascular disease with left to right shunt. Jpn J Surg 8:57-65.

Jones J, Almond CH, Snyder HM, et al. 1965. Lobar emphysema and congenital heart disease in infancy. J Thorac Cardiovasc Surg 49:1-10.

Kennedy JH, Rothman BF. 1965. The surgical treatment of congenital lobar emphysema. Surg Gynecol Obstet 121:253-60.

Lincoln JC, Stark J, Subramanian S, et al. 1971. Congenital lobar emphysema. Ann Surg 173:55-62.

Litwin SB, Rosenthal A, Fellows K. 1973. Surgical management of young infant with tetralogy of Fallot, absence of the pulmonary valve, and respiratory distress. J Thorac Cardiovasc Surg 65:552-8.

Man DW, Hamdy MH, Hendry GM, Bisset WH, Forfar JO. 1983. Congenital lobar emphysema: problems in diagnosis and management. Arch Dis Child 58:709-11.

Murray GF. 1967. Congenital lobar emphysema. Surg Gynecol Obstet 124:611-25.

Murray GF. 1971. Congenital lobar emphysema in infants. Br J Radiol 44:692-6.

Ozcelik U, Göçmen A, Kiper N, Dogru D, Dilber E, Yalçin EG. 2003. Congenital lobar emphysema: evaluation and long-term follow-up of thirty cases at a single center. Pediatr Pulmonol 35:384-91.

Pierce WS, DeParedes CG, Friedman S, Waldhausen JA. 1970. Concomitant congenital heart disease and lobar emphysema in infants: incidence, diagnosis, and operative management. Ann Surg 172:951-6.

Roghair GD. 1972. Nonoperative management of lobar emphysema lobar emphysema. Radiology 102:125-7.

Roguin N, Peleg H, Lemer J, et al. 1980. The value of cardiac catheterization and cineangiography in infantile lobar emphysema. Pediatr Radiol 10:71-4.

Rudolph AM. 1969. Commentary: pulmonary complications of congenitally heart disease. Pediatrics 43:757.

Stigers KB, Woodring JH, Konga JF. 1992. The clinical and imaging spectrum of findings in patients with congenital lobar emphysema. Pediatr Pulmonol 14:160-70.

Stranger P, Lucas PV, Edwards JE. 1969. Anatomic factors causing respiratory distress in acyanotic congenitally cardiac disease: special reference to bronchial obstruction. Pediatrics 43:760-9.