plasty was performed with a 9F-Meditech 15-mm balloon catheter. The dilations were performed three times with inflation pressure of 75 psi. The pressure gradient across the left outflow tract was decreased to 20 mm Hg. No aortic regurgitation occurred. As in case 1, 2D-echoangiography showed the image of the membrane floating in the left ventricular outflow tract. Doppler evaluation nine months after balloon dilation revealed stable reduction of the transaortic gradient.

DISCUSSION

Acquired membranous subaortic stenosis after surgical repair of cardiac malformations, including ventricular septal defect, is an uncommon event. We have recently encountered this complication in one child (case 2) in whom we performed a balloon dilation of the postsurgical subaortic obstruction; case 1 showed residual postsurgical obstruction that was treated in the same way. Percutaneous balloon dilation of discrete subaortic stenosis has been reported effective in alleviating the obstruction. Because patients who undergo surgical transection of subaortic membrane may develop recurrent obstruction requiring reoperation, balloon dilation can be considered a helpful palliative procedure.

We extended this technique to acquired subaortic stenosis with the purpose of avoiding or delaying a further surgical procedure on cardiopulmonary bypass in children who had undergone more than one surgical procedure. A significant reduction of the transmembrane pressure gradient to a nonsurgical value was achieved.

Doppler follow-up showed no acquired aortic insufficiency and a persistent decrease of the gradient achieved by balloon dilation; the technique appears to be safe and effective in reducing subaortic obstruction.

REFERENCES


Pulmonary Function Tests in Bronchopleural Fistula

Johny R. P. Alencherry, M.D.; Terrence Nigan, M.D.; and Ramesh M. Shah, M.D., F.C.C.P.

A 53-year-old white man underwent a left pneumonectomy for alveolar cell carcinoma. His postoperative course was complicated by pneumonia. At a follow-up clinic visit, the patient complained of a "roaring sound" during respiration. A follow-up PFT did not show the expected loss of volume (nitrogen washout) from a preoperative PFT, suggesting a bronchopleural fistula. A chest x-ray film and xenon lung scan confirmed the diagnosis. The fistula was surgically repaired.

(CHEST 1991; 100:582-84)

CASE REPORT

A 53-year-old white man was admitted to the hospital for resection of a mass in the left upper lobe of the lung that was found on a routine chest x-ray film. After a negative bronchoscopy, a diagnosis of squamous cell carcinoma was made by CT-guided needle aspiration biopsy at another hospital. Recently, he had hemoptysis. Thirteen years earlier the patient had had tuberculosis, which was adequately treated. He was a 40 pack-year smoker. Physical examination disclosed no abnormalities. A chest CT scan showed a 7-cm mass of the left upper lobe of the lung, with possible left hilar node enlargement. Repeat bronchoscopy was negative. Preoperative PFT showed a TLC of 7.39 L (113 percent predicted); FBC of 5.15 L (130 percent) by nitrogen washout; and an FEV, = 2.35 L (90 percent). Split perfusion lung scan showed 46 percent left and 54 percent right perfusion. There were no indications of distant metastases.

An exploratory operation was performed on the patient, and a left pneumonectomy was done. He was exubtated soon after surgery and although he developed and was treated for pneumonia of the right lower lobe, he did not require reintubation. He recovered uneventfully and fluid began to fill the left side of the chest as expected. However, just prior to discharge a drop in the fluid level was noted by the Radiology Department.

Because of a positive hilar node, he had postoperative radiation beginning 13 weeks after surgery. He received no corticosteroids. One month after radiation therapy was begun and two months after surgery, he was seen in the clinic complaining of a peculiar sound on inspiration, which he had noted before radiation treatment began. His chest x-ray film showed complete absence of fluid in the left side of the chest and a bronchopleural fistula was suspected. Pulmonary function testing had been done immediately before being seen in the clinic. It showed a TLC of 6.48 (100 percent); FBC, 4.67 (120 percent) (nitrogen washout); and FEV, 1.68 (50 percent). Auscultation showed a high-pitched distant sound on inspiration and expiration. His FFT results were felt to be consistent with a bronchopleural fistula, which had allowed measurement, by nitrogen washout, of gas in the left chest. Body plethysmography

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Pulmonary Function Tests in Bronchopleural Fistula (Alencherry, Fegan, Shah)
confirmed the TLC and FRC measurements as that of both lungs. A lung scan using technetium-labelled sulfur colloid was negative. A lung scan using xenon 133 was thought to be diagnostic of a small bronchopleural fistula, showing no uptake in the left side of the chest on a single breath, equal uptake on both sides with rebreathing and retention in the left side of the chest on washout. A CT scan of the stump was suspected of being but not diagnostic of a fistula. Bronchoscopy was not done, to decrease the chance of infecting the space. A thoracotomy was done with closure of a bronchopleural fistula with an uneventful postoperative course. No further radiation therapy was given. Follow-up chest x-ray films showed complete filling of the left side of the chest with fluid. Follow-up PFT done three months after surgery showed the TLC to be 4.85 (75 percent); FRC, 3.85 (96 percent) (nitrogen washout); and FEV1, 1.42 (42 percent).

**DISCUSSION**

This case illustrates an example of lung volume measurement with nitrogen washout detecting a bronchopleural fistula. This interesting result would have been expected using helium dilution techniques as well. The finding of an unchanged TLC and FRC from preoperative values is a completely unexpected finding in the setting of a pneumonectomy, even considering mediastinal shift.

The clinical features of this case made bronchopleural fistula a near certainty. In some cases, however, this may not be so obvious. The expected changes after pneumonectomy include progressive filling of the vacant chest with fluid, due to a net negative gas pressure around the capillaries and a net positive fluid onotic pressure in the capillaries, and a shift of the mediastinum to the operated side, and eventual opacification of the operated side.1-3 Any deviation from this suggests a bronchopleural fistula. A

The techniques used in this case to confirm the fistula are customary. The superiority of xenon 133 over sulfur colloid is expected considering the particle size differences.1-4 There have been cases where a bronchopleural fistula was almost certainly present, but was not detectable by xenon 133. No comparisons of detection by xenon 133 and nitrogen washout TLC have been published. A perfusion scan was not done as no lung parenchyma remained on the operated side.

When only a small amount of gas is present in the chest cavity after pneumonectomy, the use of nitrogen washout to detect a bronchopleural fistula would almost certainly be of little utility. However, as in this case, where a large amount of gas is present and a fistula is suspected, TLC measurement by nitrogen washout may complement the usual techniques for detection of a bronchopleural fistula. It remains to be established whether or not a fistula that is not detectable by xenon 133 would be detectable by nitrogen washout. The method is noninvasive, readily available and relatively inexpensive.

**REFERENCES**


2 Steiger MD, Wilson RF. Management of bronchopleural fistula

**Table 1—Pulmonary Function Test Results**

<table>
<thead>
<tr>
<th></th>
<th>PFT Prior to Surgery</th>
<th>PFT 6 Weeks Post-Surgery</th>
<th>PFT after Repair of Bronchopleural Fistula 2 Months Later</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC, L</td>
<td>3.94 (85)</td>
<td>3.37 (73)</td>
<td>2.26 (49)</td>
</tr>
<tr>
<td>FEV1/FVC</td>
<td>59</td>
<td>50</td>
<td>63</td>
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<tr>
<td>FEV1, L</td>
<td>2.35</td>
<td>1.68 (50)</td>
<td>1.42 (42)</td>
</tr>
<tr>
<td>TLC, L</td>
<td>7.39 (113)</td>
<td>6.48 (100)</td>
<td>4.85 (75)</td>
</tr>
<tr>
<td>RV, L</td>
<td>3.45 (156)</td>
<td>3.11 (141)</td>
<td>2.59 (118)</td>
</tr>
<tr>
<td>FRC, L</td>
<td>5.12 (130)</td>
<td>4.67 (120)</td>
<td>3.85 (96)</td>
</tr>
</tbody>
</table>

*Percent predicted values in parentheses.

Plasmapheresis in a Case of Eosinophilia-Myalgia Syndrome with Ascending Polyneuropathy

Emily Nolfo, M.D.; Vance Wright-Browne, M.B., B.S.; Michael Therrien, M.D., F.C.C.P.; Anthony Ardolino, M.D.; and Zachary Macinski, M.D.

Eosinophilia-myalgia syndrome complicated by ascending polyneuropathy in a 40-year-old woman is described. High-dose intravenous steroids had no beneficial effect on the clinical course. Dramatic and rapid clinical improvement occurred with the use of plasmapheresis. The use of this therapeutic modality should be considered in patients with a similar clinical presentation. (Chest 1991; 100:554)

The eosinophilia-myalgia syndrome is a newly described condition associated with the ingestion of L-tryptophan.1 We describe a patient with eosinophilia-myalgia syndrome and ascending polyneuropathy who showed rapid clinical recovery following treatment with plasmapheresis.

CASE REPORT

The patient was a 40-year-old black woman with a history of mental retardation. Three weeks prior to admission she was seen for complaints of dyspnea and malaise. She was thought to have an upper respiratory tract infection, and a course of antibiotics was prescribed.

Several days later she presented with no improvement in her symptoms. Physical examination was unremarkable except for a rectal temperature of 39.1°C. The chest x-ray film showed no abnormalities. The leukocyte count was 22.0 × 10^9 cells/L. The differential cell count revealed 52 percent eosinophils. On August 16, 1989 (three months before the onset of symptoms), L-tryptophan at a dose of 1 g three times a day had been started for treatment of combative premenstrual behavior. In view of the recent institution of L-tryptophan therapy, eosinophilia-myalgia syndrome was suspected. She was admitted to the hospital on November 8, 1989, and the L-tryptophan was discontinued.

Hydrocortisone, 50 mg, administered intravenously every 4 h was begun. Over the next several days, she developed an ascending flaccid paralysis and became ventilator-dependent. An electrogram and nerve conduction studies showed widespread denervation polyneuropathy and low compound action potentials, consistent with Guillain-Barre syndrome. Three serial lumbar punctures done at one-week intervals showed normal cerebrospinal fluid protein.

An empiric trial of plasmapheresis three times a week was begun on the tenth hospital day. On the 17th hospital day, the first signs of neurologic improvement were noted. The patient was soon extubated. On the 29th hospital day, she had grade 4/5 strength in the upper extremities and grade 3/5 strength in the lower extremities. Plasmapheresis was discontinued on the 33rd hospital day. She had a bilateral foot drop. She was discharged 104 days after admission and is now doing well.

DISCUSSION

The etiology of eosinophilia-myalgia syndrome remains controversial. Initial epidemiologic investigation focused on the identification of a contaminant.56 Retrospective epidemiologic studies have identified a significant association of the syndrome with a specific retail lot of L-tryptophan from a single Japanese manufacturer.45 Ascending polyneuropathy complicating the syndrome previously has been described.6 One such patient was treated with plasmapheresis and relapsed after it was discontinued. She did not respond to a second course of therapy.6 The precise clinical parameters prompting initiation and cessation of plasmapheresis were not discussed in this case. We can only postulate that the successful outcome in our case was due to early initiation of therapy and continued treatment for a prolonged period of time. All reported deaths from eosinophilia-myalgia syndrome have been associated with ascending polyneuropathy.

Plasmapheresis may either replace a depleted plasma constituent or remove an abnormal one.7 It is likely that the removal of an abnormal constituent (contaminant, toxic metabolites, immune complexes, or mediators of inflammation) was the reason for the patient's improvement. The 1-week lag period separating the start of plasmapheresis and the improvement of clinical symptoms could be due to the gradual removal of substances deposited in tissues.

We suggest that plasmapheresis be considered in patients with eosinophilia-myalgia syndrome complicated by ascending polyneuropathy. We also urge that further investigation of this treatment modality be pursued.

REFERENCES


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