Embolectomy for Chronic Pulmonary Embolism and Hypertension

Case Report and Review of the Problem

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SUMMARY

The case of a 31-year-old Negro who had had two admissions to the hospital at which pulmonary emboli were diagnosed is reported. On his third admission with chest pain, hemoptysis, and dyspnea, chest roentgenograms revealed increased pulmonary vascular markings on the right and a prominent pulmonary artery which was sharply cut off. ECG changes were typical of pulmonary artery obstruction and pulmonary scan revealed a small perfusion defect in the lower lobe of the right lung and complete absence of perfusion in the left lung. The left pulmonary artery was occluded. An organized thrombus was removed from the left pulmonary artery and from most of its distal branches. Good back flow ensued, and 10 months after operation the patient was well except for some edema of the leg.

A discussion of the problems and indications for pulmonary embolectomy is offered.

Massive embolic occlusion of the pulmonary arteries is usually an acute phenomenon and presents dramatic clinical manifestations. Recently considerable emphasis has been placed upon the role of pulmonary embolectomy as a lifesaving emergency measure in these circumstances. Chronic cor pulmonale from repeated pulmonary emboli is also a well-established disorder, and despite its grave prognosis, scant attention has been given surgical treatment of this entity. It is thought that chronic pulmonary embolism and hypertension are more common than generally recognized, and it is now feasible to establish an accurate preoperative diagnosis and consider a surgical approach in the management.

In 1950 Carroll reported on the first patient in whom a diagnosis of chronic occlusion of a pulmonary artery was made before death, and Hollister and Cull first suggested the operative removal of thromboembolic material from a chronically occluded pulmonary artery. There have been several direct attempts to restore patency in a pulmonary artery chronically occluded by an embolus with limited success in restoring blood flow to part or all of the involved lung (Scott, S. M.: personal communication to authors). The purpose of the present report is to record observations in the objective preoperative assessment of this disorder and to place emphasis on important features of surgical correction and postoperative evaluation.

Clinical Observations

The following patient was studied extensively preoperatively and considered to be an appropriate candidate for surgical treatment. The objective studies performed with documentation of the postoperative changes indicate the gratifying result which can be achieved by aggressive surgical management.

Report of Case

A 31-year-old man was first admitted to the Duke University Medical Center in May 1967...
with chest pain, hemoptysis, dyspnea, and clinical evidence of thrombophlebitis. A phlebogram demonstrated the presence of thrombi in the leg veins and a pulmonary scan showed a significant decrease in perfusion of both lungs consistent with multiple pulmonary emboli. The patient was treated with heparin and after the thrombophlebitis had subsided was discharged to continue treatment with coumadin. One month later he was readmitted with hemoptysis and dyspnea, and a pulmonary scan showed evidence of additional pulmonary emboli. A suture screen was placed in the inferior vena cava for prevention of further emboli, and he was again discharged on coumadin. In early June 1968, he again had sudden onset of chest pain, hemoptysis, and dyspnea and was readmitted. On examination the resting respiratory rate was 24/min, the pulse was 82/min and regular, and the temperature and blood pressure were normal. The neck veins were distended 6 cm above the clavicle with the patient reclining at 45°. The lungs were clear and no murmurs were heard. The second pulmonary sound was palpable. There was a fixed split second heart sound and P₂ was louder than A₂. An S₃ gallop was heard which increased in intensity with inspiration. The liver was palpable 2 cm below the right costal margin but did not pulsate and was not tender. There was no evidence of edema or tenderness of the lower extremities. The hematocrit was 53%. The chest film revealed increased pulmonary vascular markings on the right (fig. 1A). The pulmonary artery on the left was prominent with a sharp cutoff, and the peripheral vascular markings were decreased. The left hemithorax was more radiolucent than the right. The changes in the electrocardiogram were compatible with pulmonary artery obstruction (fig. 2). The mean QRS axis was shifted to the right (+135°) with a shift of the initial forces leftward with a rotation of the T-wave leftward and posteriorly as seen in right ventricular strain. An incomplete right bundle-branch block was present with P waves suggestive of P pulmonale. A pulmonary scan was done which showed a moderate-sized perfusion defect in the right lower lobe and a complete absence of perfusion in the left lung (fig. 1B). An inferior vena cavaogram was performed with biplane cineangiography showing that the lumen of the inferior vena cava was not narrowed as might be suspected from the caval operation. The left femoral vein was occluded with thrombus. On July 2, 1968, the inferior vena cava was ligated immediately below the renal veins in an effort to prevent further pulmonary emboli in a patient repeatedly resistant to anticoagulant therapy. The suture screen previously placed in the vena cava was found to be disrupted. Postoperatively the patient was heparinized and later was given coumadin. Right heart catheterization was performed; the pulmonary arterial pressure was 75/20 mm Hg (mean, 42) and the right ventricular pressure was 85/4 mm Hg (mean, 30). Pulmonary arteriograms showed complete occlusion of the left pulmonary artery at the bifurcation of the main pulmonary artery and a decrease in perfusion of the right lower lobe artery (fig. 1C). Pulmonary function studies preoperatively (table 1) showed a severe ventilation perfusion imbalance with an estimated minimum of 1,135 ml of lung tissue ventilated but not perfused. The arterial Po₂ was 65 and Pco₂ 31 mm Hg. A diagnosis of chronic pulmonary

**Figure 1**

(A) Preoperative roentgenogram of thorax showing increased vascular markings on the right side. The left main pulmonary artery is prominent with a sharp cutoff and decreased vascular markings in the left hilum. The left hemithorax is more radiolucent than the right.

(B) The lung scan shows some decrease in perfusion in the right mid-lung field and in the right base. There is no perfusion in the left lung.

(C) Pulmonary arteriogram shows a complete occlusion of the left pulmonary artery and decreased perfusion in the right lower lobe.
PULMONARY EMBOLECTOMY

Figure 2

Preoperative ECG shows a mean QRS of +135°. There is shift of the initial forces leftward with a rotation of the T wave leftward and posteriorly as seen in right ventricular strain. An incomplete right bundle-branch block is present and the P waves are suggestive of P pulmonale.

Table 1

Pulmonary Function Data Before and After Operation

<table>
<thead>
<tr>
<th></th>
<th>Calculated normal</th>
<th>Preop</th>
<th>2 weeks PO</th>
<th>14 weeks PO</th>
<th>9 mo PO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital capacity (L)</td>
<td>4.63</td>
<td>3.01</td>
<td>2.42</td>
<td>3.29</td>
<td>3.12</td>
</tr>
<tr>
<td>Residual volume (L)</td>
<td>1.45</td>
<td>2.81</td>
<td>1.70</td>
<td>1.66</td>
<td>1.43</td>
</tr>
<tr>
<td>Total lung capacity (L)</td>
<td>6.08</td>
<td>5.82</td>
<td>4.12</td>
<td>4.95</td>
<td>4.64</td>
</tr>
<tr>
<td>FEV1 (L/sec)</td>
<td>3.82</td>
<td>2.38</td>
<td>1.92</td>
<td>2.45</td>
<td>2.62</td>
</tr>
<tr>
<td>Paco2 (mmHg)</td>
<td>-</td>
<td>31</td>
<td>33</td>
<td>34</td>
<td>38</td>
</tr>
<tr>
<td>PaO2 (mmHg)</td>
<td>-</td>
<td>65</td>
<td>77</td>
<td>81</td>
<td>76</td>
</tr>
<tr>
<td>Arterial pH</td>
<td>-</td>
<td>7.50</td>
<td>7.44</td>
<td>7.42</td>
<td>7.41</td>
</tr>
<tr>
<td>Tidal volume (ml)</td>
<td>-</td>
<td>672</td>
<td>586</td>
<td>540</td>
<td>498</td>
</tr>
<tr>
<td>A-a O2 gradient (mmHg)</td>
<td>-</td>
<td>51</td>
<td>39</td>
<td>32</td>
<td>30</td>
</tr>
<tr>
<td>Physiologic dead space (ml)</td>
<td>150</td>
<td>295</td>
<td>207</td>
<td>191</td>
<td>185</td>
</tr>
<tr>
<td>Pulmonary artery pressure</td>
<td>-</td>
<td>75/20</td>
<td>40/30 (35)</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

|                              |                   |       |            |             |         |
| Physiologic dead space (ml)  |                   |       |            |             |         |
| (mm Hg)                      |                   |       |            |             |         |

Abbreviations: FEV = forced expiratory volume; Paco2 = partial pressure of CO2 in arterial blood; PaO2 = partial pressure of O2 in arterial blood; A-a O2 gradient = alveolar to arterial O2 gradient; PO = post operative.

Embolism with pulmonary hypertension and cor pulmonale was made.

On July 18, 1968, more than a year after the original embolism and 6 weeks after the most recent embolic episode, pulmonary embolectomy was performed employing extracorporeal circulation. The heart and the left pulmonary artery were exposed through a left anterolateral thoracotomy in the fourth intercostal space. On opening the pericardium it was found that the right side of the heart was dilated and hypertrophied. The main pulmonary artery was tense and the left pulmonary artery was firm and pulseless. There was dense inflammatory reaction about the left pulmonary artery, particularly in the extrapericardial portion. Cardiopulmonary bypass was established by draining the venous blood through a cannula in the outflow tract of the right ventricle with return of oxygenated blood through a cannula into the left femoral
from the distal branches of the left pulmonary artery, a point of great importance. The arterioto- mies were closed and cardiopulmonary bypass was discontinued. The pressure in the main pulmonary artery was considerably lower than that preoperatively. After the outflow cannula was removed, the pulmonary arterial pressure was 40/30 mm Hg. The pericardium and left pleural cavity were drained and the incision was closed. Microscopically the embolus was an old and organized thrombus with chronic inflammatory cell infiltration (fig. 4). By history the first episode had occurred more than a year previously and the last embolic episode occurred at least 36 days prior to embolectomy.

The postoperative course was uneventful and the patient noted the absence of dyspnea and an early increase of exercise tolerance. One week postoperatively the blood gas values had improved considerably. A pulmonary scan and arteriogram showed perfusion to most of the left lung (fig. 5). Fourteen weeks postoperatively there was further improvement in the blood gas values and the lung scan showed a near normal perfusion (fig. 6). Postoperatively pulmonary function studies demonstrated a progressive decrease in the ventilation perfusion imbalance (table 1). The patient was maintained on coumadin and walked early with elastic stockings. Dyspnea was present only with strenuous exertion. The patient has been followed for an additional year and remains well except for edema of the lower extremities late in the day or in the absence of elastic support, as might be expected following ligation of the inferior vena cava. The electrocardiogram recorded 9 months postoperatively is normal (fig. 7).

**Discussion**

Chronic pulmonary embolism has been clearly recognized as a cause of pulmonary hypertension and chronic cor pulmonale. With the advent of newer surgical technics and particularly of extracorporeal circulation, embolectomy for acute massive embolism has become feasible with demonstration that pulmonary blood flow can be reestablished. In the recent past these observations have been extended to a limited degree in patients with chronic pulmonary embolism.

The incidence of chronic pulmonary embolism and cor pulmonale is not known but is thought to be considerably greater than is generally recognized. Prior to 1941 a total of 100 cases had been published, and Hollister and Cull reported an additional 100 cases.
Figure 4

Photomicrograph of the removed specimen shows organization with granulation tissue and a chronic inflammatory cell infiltrate.

Figure 5

(A) The chest roentgenogram on the fifth postoperative day shows some minor atelectasis and pleural reaction in the left mid-lung field and around the pulmonary artery. (B) The lung scan shows perfusion on the left side. (C) The pulmonary arteriogram shows perfusion through the left pulmonary artery.

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Figure 6
(A) The chest roentgenogram in the eighth postoperative week is normal except for the remaining prominence of the pulmonary artery in the left hilum. (B) The perfusion scan is almost normal except for a small defect in the right lower lobe. (C) The pulmonary arteriogram shows that blood flow is present on the left side. Perfusion is still absent in some branches of the right lower lobe and in the lingular branches of the left upper lobe.

Figure 7
The ECG, 9 months postoperatively, has returned to normal.

from 1941 to 1945. In all of these, however, the diagnosis was made post mortem. In 1950 Carroll described five patients in whom the diagnosis of chronic pulmonary arterial obstruction was established by cardiac catheterization. The diagnosis has been made with increasing frequency since that report and is in part a result of the more widespread use of pulmonary scanning and arteriography. The clinical manifestations of chronic pulmonary embolism are often characteristic, and the diagnosis is being made with increasing frequency.1-22 Lung scanning with macroaggregated radioiodinated human serum al-
Table 2

**Operations for Removal of Chronic Pulmonary Emboli**

<table>
<thead>
<tr>
<th>Author</th>
<th>Date of operation</th>
<th>Site of obstruction</th>
<th>Result</th>
<th>Backbleeding from distal pulmonary artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hurwitt et al.³</td>
<td>1957</td>
<td>Bilateral</td>
<td>Died</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Allison et al.⁴</td>
<td>1958</td>
<td>Bilateral</td>
<td>Improved</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Snyder et al.⁵</td>
<td>1961</td>
<td>Right PA</td>
<td>Improved</td>
<td>Good</td>
</tr>
<tr>
<td>Houk et al.⁶</td>
<td>1961</td>
<td>Bilateral</td>
<td>Improved</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Moser et al.⁷</td>
<td>1962</td>
<td>Right PA</td>
<td>Improved</td>
<td>Good</td>
</tr>
<tr>
<td>Prater et al.⁸</td>
<td>1963</td>
<td>Bilateral</td>
<td>Improved</td>
<td>Good</td>
</tr>
<tr>
<td>Moser et al.⁷</td>
<td>1964</td>
<td>Bilateral</td>
<td>Died</td>
<td>None</td>
</tr>
<tr>
<td>Scannell (Castleman⁹)</td>
<td>1964</td>
<td>Bilateral</td>
<td>Died</td>
<td>None</td>
</tr>
<tr>
<td>Makey et al.¹⁰</td>
<td>1965</td>
<td>Left PA</td>
<td>Improved</td>
<td>Good</td>
</tr>
<tr>
<td>Scott (personal communication)</td>
<td>1965</td>
<td>Bilateral</td>
<td>Died</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Nash et al.¹¹</td>
<td>1966</td>
<td>Left PA</td>
<td>Improved</td>
<td>Good</td>
</tr>
<tr>
<td>Present case</td>
<td>1968</td>
<td>Bilateral</td>
<td>Improved</td>
<td>Good</td>
</tr>
</tbody>
</table>

Bumin²¹ is a simple and effective screening procedure for establishing the diagnosis for most patients. Cardiac catheterization and pulmonary arteriography provide additional information regarding the extent of the pulmonary hypertension and the site of arterial occlusion.

Since current clinical and experimental evidence suggests that pulmonary embolism is primarily a mechanical problem,⁷ 23–25 the operative removal of emboli from the pulmonary vessels is a logical form of treatment. In the literature, 12 patients with chronic pulmonary embolism have been managed by embolectomy and of these, eight were successful. It appears clear that appreciable back bleeding at the time of embolectomy is of great importance and it was present in six of the eight successful cases reported. Back bleeding was not mentioned in two of the four cases in which death occurred,⁴ and it was not present in two.⁷ ⁹ In one of the latter, autopsy revealed that the pulmonary artery was patent distal to the obstruction. Thus, emphasis should be placed upon the importance of back bleeding following thrombectomy as a favorable sign representing collateral flow from bronchial vessels. Indeed, the presence of these collaterals is probably responsible for halting propagation of thrombosis distal to the embolus and maintaining patency of the small pulmonary arteries.

The encouraging results obtained in the present report as well as those noted in the literature (table 2) appear to establish a firm role for operative treatment in selected cases of chronic pulmonary embolism and hypertension. This is particularly important in view of the poor prognosis of patients treated nonoperatively. Firm indications should be present for surgical intervention. The patient clearly should have considerable dyspnea and preferably no serious additional cardiac or respiratory problems. Studies should demonstrate occlusion of the right or left main pulmonary artery and not solely occlusion of the smaller distal branches.

Emphasis should also be placed upon several points in the surgical procedure. The operative approach must be individualized by the location of the arterial occlusion. It is important that the arteriotomy be placed to allow exposure of the secondary branches because of the firm attachment of the emboli to the intima. This attachment precludes easy removal of the thromboembolic material by suction, as is frequently possible in acute embolism. Fortunately, there is sufficient organization of the chronic embolus to allow extraction by traction on even the most distal portion of the emboli. Care must be taken to avoid disruption of the elastic distal fragments and resultant retraction into the peripheral branch of the artery. A balloon embolectomy catheter is generally ineffective in removing the distal material. Continuation of anticoagu-
lation postoperatively is thought important in obtaining the most favorable results.

References
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