misleading.

Paralytic disease associated with echovirus 2 infection has previously been reported as a complication of aseptic meningitis due to echovirus 2 prime, mostly affecting infants and one adolescent who also had pleurodynia. Only one report concerning neurotoxic effects of this virus mentions convulsions, but the ages of the patients are not given.

Echovirus 2 has been specifically associated with myocardial disease in infants, and electrocardiographic documentation of this involvement consisting of ST depressions and T wave changes was obtained. In another outbreak of nosocomial infection involving this virus, an infant was described with significant sinus bradycardia and other cardiac complications necessitating digitalization. The present thinking about enterovirus myocardiopathy has been summarized by Lerner.

Of additional conceptual significance is the recently presented evidence that ECGs and x-ray films may be relatively insensitive indicators of cardiotoxicity due to cellular damage, as compared with measurement of systolic time intervals. This was found for alcoholic myocardiopathy and for the myocardiopathy due to chemotherapeutic agents, in particular adriamycin. Myocardial damage of very significant degree in our patient was detected by this method long after the ECG became normal, and the changes could be followed serially and quantitatively.

In noninvasive studies of cardiac patients, high correlations have been found to exist between PEP/LVET ratios and diminished cardiac output, stroke volume, and ejection fraction. Detection and quantitation of myocardial damage is important in view of the experimental and clinically demonstrated untoward effect of exercise in this cardiomyopathy. Also, following previously established recommendations, cortisone administration was prohibited, the patient was warned of alcohol toxicity, and the desirability of avoiding pregnancy was stressed. Continued follow-up of such patients by this method is suggested to determine development of chronic myocardiopathy.

REFERENCES


Anomalous Communication of the Left Coronary Artery with a Peripheral Branch of the Right Pulmonary Artery*

Roberto J. Macchi, M.D.; Ramon A. Fabregas, M.D.; Hector O. Chianelli, M.D.; Juan C. Bussonlino Bourdet, M.D.; Oswaldo Lhez, M.D.; and Ricardo Stagnaro, M.D.

A 64-year-old man had a history of two myocardial infarctions and recurrent episodes of paroxysmal ventricular tachycardia and hemoptysis. Cineangiographic studies showed an anomalous vessel connecting the left coronary artery with a peripheral branch of the right pulmonary artery. Although several coronary obstructive lesions and abnormalities in the distribution of the circumflex branches were found, the anomaly apparently produced a real "pulmonary steal" and, thereby, a worsening of the coronary insufficiency. We believe that this is the first reported case in the literature with such a coronary-pulmonary fistula. The angiographic findings are presented in detail, and the possible factors in the development of myocardial ischemia are discussed.

There have been reports of several cases of abnormal connections between a coronary artery and other sections of the cardiovascular system, namely, a cardiac chamber or the pulmonary trunk. Nevertheless, on a review of the literature, we were unable to find a case in which there was an abnormal communication between the left coronary artery and a peripheral branch of the

*From the Cardiovascular Laboratory and the Division of Cardiology, Clinica Modelo, Moron, Buenos Aires, and the Cardiology Division, Department of Medicine, Albany Medical College and Veterans Administration Hospital, Albany, NY.

Reprint requests: Dr. Fabregas, VA Hospital, Albany, New York 12208

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pulmonary artery. The purpose of this report is to describe such a case.

**CASE REPORT**

In November 1973, a 64-year-old white man was admitted to the clinic because of sudden onset of syncope, dyspnea, and marked diaphoresis. His past history revealed that at age 20 (1929), the patient experienced an episode of hemoptysis, but no definite cause was found. At age 48 (1957), the patient again had two episodes of hemoptysis which remained unexplained.

The patient carried on a normal life until March 1971, when he had two episodes of paroxysmal ventricular tachycardia and heart failure which were controlled with antiarrhythmic therapy and digitalization. An electrocardiogram at this time showed first degree atrioventricular block, complete right bundle-branch block, and left anterior hemiblock.

In October 1972, the patient developed an acute febrile pulmonary illness diagnosed as interstitial pneumonitis of the right lower lobe. In May 1973, the patient experienced epigastric pain, dyspnea, and hemoptysis. A chest x-ray film showed left ventricular enlargement, an apical deformity suggestive of a ventricular aneurysm, pulmonary vascular congestion, interlobar pleural effusion in both lungs, and increased vascularity in the right lower lobe.

The patient had to be readmitted in September 1973 because of a new episode of paroxysmal ventricular tachycardia; this was easily corrected with electrical cardioversion.

In October and November 1973, the episodes of ventricular tachycardia repeated. At this time the patient's blood pressure was 100/70 mm Hg, and the lungs had fine inspiratory rales in both bases. The point of maximal impulse was palpable at the sixth left intercostal space in the anterior axillary line; no heaves were felt, and the second sound was widely split at the pulmonary focus and remained fixed during inspiration. There was no cyanosis, no clubbing, and no peripheral edema. The hemoglobin level was 14.3 gm/100 ml, and the red blood cell count was 4,900,000/cu mm.

The patient underwent selective coronary angiographic studies, right cardiac catheterization, and pulmonary angiographic studies. There was a dominant right coronary artery which showed two areas of moderate narrowing, between 30 and 50 percent, immediately proximal and distal to a marginal branch (Fig 1). The opacification of the left coronary artery showed a normal main trunk, total occlusion at the origin of the anterior descending branch, and an unusual distribution of the circumflex artery, which had four branches. Two of these branches surrounded the base of the left atrial appendage and turned posteriorly, toward the atrioventricular sulcus, as in a normal situation (Fig 2). The other two branches of the circumflex made a turn downward and perfused the lateral wall of the left ventricle. Immediately before the origin of these circumflex branches, a subtotal segmental obstruction was visualized. There were also severe lesions in the beginning of the first diagonal. No collateral vessels were seen; however, the most remarkable finding was the visualization of a large and tortuous anomalous vessel, arising apparently from the main trunk of the left coronary artery and running dorsally behind the aorta and toward the right, ending in a pulmonary peripheral branch of the apical segment of the right inferior pulmonary lobe (Fig 2 to 4). The contrast medium was promptly washed away from the pulmonary artery.

**Figure 1.** Selective right coronary arteriogram in left anterior oblique projection showing two areas of arterial narrowing (arrows). M, Marginal branch; and PD, posterior descending branch. **Figure 2.** Selected frame of left coronary arteriogram in right anterior oblique projection showing a short main trunk, total occlusion of diagonal (D). Solid arrow indicates 50-percent occluding lesion near origin of one of circumflex (CF) atrioventricular branches. Note size and tortuosity of anomalous vessel (AV) oriented posteriorly and to right; and connecting the left coronary artery (LCA) with peripheral branch of pulmonary artery. **Figure 3.** Selected frame of left coronary arteriogram in right anterior oblique projection showing long tortuous course of anomalous vessel until it reaches peripheral branch (PAB) of right pulmonary artery.
pulmonary artery branch and then ran through the corresponding pulmonary vein. No opacification of the left atrium was seen.

Left ventricular cineangiographic studies demonstrated no mitral insufficiency but did visualize an anterior wall aneurysm and inferior wall hypokinesia. An intramural thrombus was strongly suspected. The left ventricular end-diastolic pressure was 25 mm Hg.

Right cardiac catheterization revealed increased pulmonary arterial and pulmonary wedge mean pressures (Table 1). Although the wedge pressures in both lungs were not obtained simultaneously, higher values were noted in the right lower lobe. There was no evidence of an arteriovenous shunt at the right pulmonary arterial branch level, either by oximetric data or hydrogen-inhalation curves. The oxygen saturation in all chambers was normal.

A selective pulmonary cineangiogram showed a normal arterial and venous pattern.

**DISCUSSION**

According to the reported cases of abnormal communications between the coronary arterial system and other structures, two main types of anomalies can be found. In the first type, there is an anomalous origin of one or both coronary arteries, usually the left coronary, with the involved vessel arising from the main trunk of the pulmonary artery. In the other type the coronary arteries arise from the aorta, with a branch which connects directly either with a cardiac chamber or with the pulmonary trunk. Although the vessel had a peripheral pulmonary termination in our case, it could be included in the second group of anomalies, considering the normal origin of the coronary arteries. In this group the cases of coronary-pulmonary fistulas are rare. Ogden,2 in a series of 224 cases of congenital anomalies of the coronary arteries, reported 31 cases of coronary "arteriovenous" fistulas, four of which ended in the pulmonary artery. In all of these four cases, the fistulas terminated in the pulmonary trunk.

Whether of acquired or congenital origin, the lesion of our patient, because of its size and tortuosity, suggests that the "fistula" had been present for many years. The absence of dilation in the left coronary artery proximal to the origin of the "fistula" would argue against a congenital lesion.

In the anomalous origin of one of the coronary arteries from the pulmonary trunk, it has been postulated that there is a decrease in myocardial blood flow due to preferential shunting from the normal coronary artery towards the low resistance of the anomalous coronary artery and, hence, to the pulmonary circulation. However, this physiologic mechanism is not totally applicable to our case, because of the normal origin of the left coronary artery. Our patient’s clinical course seems to support this impression, since his tolerance of the lesion was excellent almost until he entered his fifth decade and in spite of the size of the fistula and the apparent importance of the coronary-pulmonary shunt. The worsening of symptoms could be satisfactorily explained by the development of progressive coronary obstructive lesions distal to the fistula. The arterial occlusion produced an area of ischemia or infarction. The coronary obstruction also favored an increase of the "pulmonary steal," which, in turn, aggravated the myocardial ischemia and the extension of the infarction. The coronary obstruction also facilitated the formation of a ventricular wall aneurysm and the onset of heart failure. This explanation seems plausible and is partially supported by several reports of cases in which the shunting of blood from the coronary circulation produced myocardial failure.

The reason for the lack of oximetric and hydrogen-inhalation evidence for an arteriovenous shunt at the

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**Table 1—Catheterization Data**

<table>
<thead>
<tr>
<th>Site</th>
<th>Oxygen Pressure, mm Hg*</th>
<th>Oxygen Saturation, %</th>
<th>Hydrogen Inhalation**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior vena cava</td>
<td>10/2 (6)</td>
<td>72</td>
<td>—</td>
</tr>
<tr>
<td>Right atrium</td>
<td>12/4 (7)</td>
<td>74</td>
<td>—</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>58/0†</td>
<td>75</td>
<td>—</td>
</tr>
<tr>
<td>Pulmonary artery, trunk</td>
<td>55/26 (43)</td>
<td>74</td>
<td>—</td>
</tr>
<tr>
<td>Pulmonary artery, right branch</td>
<td>. . .</td>
<td>76</td>
<td>—</td>
</tr>
<tr>
<td>Pulmonary artery, left branch</td>
<td>. . .</td>
<td>75</td>
<td>—</td>
</tr>
<tr>
<td>Pulmonary artery, right lower &quot;wedge&quot;</td>
<td>. . .</td>
<td>96</td>
<td>. . .</td>
</tr>
<tr>
<td>Pulmonary artery, left lower &quot;wedge&quot;</td>
<td>. . .</td>
<td>97</td>
<td>. . .</td>
</tr>
<tr>
<td>Ascending aorta</td>
<td>117/75 (95)</td>
<td>94</td>
<td>. . .</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>117/30‡</td>
<td>94</td>
<td>. . .</td>
</tr>
</tbody>
</table>

*Mean pressures indicated by parentheses.

**Minus signs mean negative for shunt.

†End-diastolic pressure, 2.

‡End-diastolic pressure, 25.
level of the right pulmonary branch is not clear, but we think that it does not necessarily invalidate the diagnosis; similar negative findings have been reported in some other cases of coronary-pulmonary fistulas with proven left-to-right shunts. Gobel et al. reported that in their cases the shunts were small in comparison with the cardiac output and that the shunts may be difficult or impossible to detect. This explanation is not easy to accept in our case, where the shunt appeared to be quite large in the angiographic examination. For the same reason the absence of vascular murmurs in all thoracic areas was remarkable.

The finding of a higher pressure at the right inferior wedge position than in a similar localization of the left side can be questioned, since both determinations were not made simultaneously. However, the magnitude of the pressure change, and the shape of the curves suggested that it could be possible that there was a real increase of the “capillary” blood pressure in the affected lobe. This elevation was greater than the left ventricular end-diastolic pressure. This “local capillary pulmonary hypertension” could be partially responsible for the recurrent episodes of hemoptysis, since no cause other than the fistula was evident.

Because of the rarity of the lesion and the advanced obstructive lesions of the left coronary artery, any opinion regarding the best method of treatment is questionable.

REFERENCES

ANNOUNCEMENTS

Sixth Annual Course on Chest Radiology

The Fleischner Society will present the Sixth Annual Course on Chest Radiology in Zagreb, Yugoslavia, May 30-June 1. All presentations will be in English with earphone translations. Please address inquiries to Benjamin Felson, M.D., President, Fleischner Society, General Hospital, Cincinnati 45267.

International Congress on Emergency and Critical Care Medicine

The International Congress on Emergency and Critical Care Medicine, honoring the Bicentennial Anniversary of the United States of America, will be held at the William Penn Hotel, Pittsburgh, May 4-8. For details of the program, please write Dr. Peter Safar, Room 1060C Scaife Hall, University of Pittsburgh School of Medicine, Pittsburgh 15261.

Fourth Czechoslovak Pneumo-Phthisiological Congress

The Slovak Medical Society and the Slovak Pneumo-Phthisiological Society will sponsor the Fourth Czechoslovak Pneumo-Phthisiological Congress in Bratislava, May 27-29. Official language is English with earphone translations. Please address inquiries to the Congress Office.