Rupture of an Aortic Aneurysm into the Pulmonary Artery
A Case Report

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A case report of rupture of an aortic aneurysm with antemortem diagnosis and a brief review of the common clinical findings in this syndrome are presented. The case reported was studied by cardiac catheterization which, it is felt, confirmed the clinical diagnosis. Postmortem findings are given and a drawing and photograph of the specimen is shown.

THE SYNDROME of rupture of an aortic aneurysm into the pulmonary artery, though uncommon, has been reported with sufficient frequency as to constitute a fairly definite clinical entity. A review of the literature in 1943 by Nicholson revealed 81 case reports, to which he added two of his own. Ninety-one per cent of these cases occurred in males. In 85 per cent of the cases the location of the aneurysm was in the ascending aorta. It was noted that if death occurred soon after rupture, the tear was usually irregular in shape; however, if erosion was slow the aperture was usually smooth and oval. The characteristic murmur noted in most cases was a continuous one with systolic accentuation, though in some of the cases the murmur was noted as being systolic only. There was a thrill in 68 per cent of the cases, and in the majority the timing was either systolic or continuous. A collapsing pulse was demonstrated in more than half of the cases, and cyanosis or pallor was noted in 66 per cent. Edema of the extremities, dyspnea and cough were prominent symptoms. The x-ray picture usually revealed an aneurysmal sac extending to the left of the sternum with an associated dilatation of the pulmonary conus. Slight to marked cardiac enlargement was usually described. Electrocardiograms reported in six cases did not appear to be specific.

According to Nicholson, the typical clinical picture consists of a sudden onset of severe precordial pain and dyspnea after strain. Edema develops rapidly and cyanosis or pallor is often seen. At the onset euphoria and oliguria may be present. The extremities are frequently cold to the touch. The patient develops paroxysms of coughing with expectoration; palpitation may be a prominent feature. On examination, the patient usually appears acutely ill and exhibits moderate dyspnea. Moderate pallor or cyanosis and edema are usually observed. An intense purring thrill, either systolic, diastolic or both is usually palpable over the precordium at the pulmonic area. On auscultation a typical crescendo-decrescendo harsh murmur can usually be heard. The usual signs of aortic regurgitation are present.

It was noted by Nicholson that only four of the cases reviewed had been diagnosed ante mortem; however, he presented two case reports of his own wherein the diagnosis had been made before death.

In 1950, Klein and Porter presented a case report wherein the patient survived 14 months after onset. Of special interest in this case report were the catheterization studies carried out during the hospitalization of the patient. In summary, the results of these studies revealed an increased pressure in the right ventricle and pulmonary artery and increased oxygen saturation of blood in the pulmonary artery. Catheterization in this case was considered to be an important diagnostic aid. A correct antemortem clinical diagnosis was made in this case which was definitely confirmed by catheterization studies.

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In view of the infrequent antemortem diagnosis made in these cases and in view of the valuable information obtained by heart catheterization, it was felt that a case report wherein a correct antemortem diagnosis had been made would be of considerable interest.

CASE REPORT

J. W., a 28 year old Negro man, was admitted to the University Hospital on Oct. 25, 1949, with the chief complaint of shortness of breath and swelling of the face, feet and ankles.

History of present illness revealed that the patient had to stop work about five weeks before admission because of a cough and a constant choking sensation. Dyspnea increased, and two weeks prior to admission the patient noted onset of swelling of the face, abdomen and lower extremities. On this same date he noted that the shortness of breath had increased, and he began to experience orthopnea at night. Because of increasing dyspnea the patient was forced to go to bed about one week prior to admission.

Past History. In 1936 the patient had had lymphogranuloma venerum. For the past five years the patient noted that he seemed to keep a cold most of the time, usually accompanied by an annoying cough. A positive serologic reaction was noted in 1943 and was followed by two years of antisyphilitic therapy with bismuth and arsenic preparations. Repeat serologic test in 1945 was again positive and the patient received six additional months of antisyphilitic therapy. About one year prior to admission he developed a sudden pain in his left chest with radiation to the posterior thorax. At that time he had the sensation that something had "snapped" in his chest. Following the attack he remained in bed for four days and experienced a suffocating sensation most of the time. During this period he also noted marked palpitation and afterwards experienced difficulty sleeping because of frequent sensations of suffocating after going to bed.

On admission examination revealed a well developed and well nourished Negro man appearing acutely ill and exhibiting orthopnea. The blood pressure was 145/60. The pulse rate was 90, and of Corrigan type. There was obvious facial and peri-orbital edema; the neck veins were distended. The left cardiac border was at the anterior axillary line, the right border along the right margin of the sternum. A systolic thrill was palpable in the third left intercostal space 4 cm. from the midsternal line. In this same area there was a loud, harsh, blowing systolic murmur and a fainter diastolic murmur. The murmur was of the crescendo-decrescendo type. The murmur tended to diminish in intensity if auscultation was performed at any area away from the third left intercostal space. Medium moist rales were noted at both bases. The liver was enlarged three fingerbreadths below the right costal margin, and a hepatojugular reflex was present. Moderate ascites was also present. Three plus presacral edema and 3 plus edema of the lower extremities were present.

The red blood cells numbered 4,660,000; the white blood cells, 5,300. Hemoglobin was 14 Gm. Neutrophils were 61 per cent, lymphocytes 30 per cent, monocytes 4 per cent, eosinophils 5 per cent. Corrected sedimentation rate was 14 mm. in one hour. Preparations for sickling were negative. Nonprotein nitrogen was 29 mg. per 100 cc., sugar 105 mg. per 100 cc. and chlorides 460 mg. per 100 cc. The blood Kahn test was negative. Total proteins were 6.15 Gm. per 100 cc.; albumin 3.3 Gm. per 100 cc., globulin 2.85 Gm. per 100 cc. Spinal fluid examination revealed total protein of 32 mg. per 100 cc. with a normal colloidal gold curve. Quantitative Wasserman on spinal fluid was negative for 0.1, 0.25, and 0.5 cc. of fluid, but was positive for 1 cc. of fluid. On admission the electrocardiogram revealed sinus tachycardia with incomplete right bundle branch block. Urinalysis revealed a 2 plus albuminuria with a specific gravity of 1.010. X-ray of the chest (fig. 1) revealed considerable cardiac enlargement with pulmonary congestion bilaterally. Unusual enlargement of the pulmonary conus area was noted.

The admission clinical diagnosis was (1) syphilitic heart disease with syphilitic aortitis and aortic insufficiency; (2) congestive heart failure; (3) left hydrothorax.

On October 26 venous pressure was 300 mm. H2O. The circulation time, arm to tongue, with Decholin was 25 seconds. The patient was started on digitalis.

FIG. 1. X-ray film of the patient's chest.
and diuretics in addition to oxygen and a low sodium diet. Some improvement was obtained; however, the patient continued to have medium rates at both bases and to experience moderate dyspnea and at times orthopnea. It was the impression of two observers, after fluoroscopy of the chest, that the diagnosis was probably patent ductus arteriosus. However, after a review of the history and findings, it was the impression of one of the authors (V. P. S.) that the diagnosis in this case was rupture of a syphilitic aortic aneurysm into the pulmonary artery. In view of this impression the patient was scheduled for cardiac catheterization, and on Nov. 8, 1949, cardiac catheterization was performed by Doctors R. G. Ellison, W. F. Hamilton, Jr. and W. F. Ham-

Table 1.—Physiologic Observations Obtained from Catheterization Studies

<table>
<thead>
<tr>
<th>Position Catheter</th>
<th>Oxygen Content</th>
<th>% Saturated</th>
<th>Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. P.A. (right)</td>
<td>14.79 Vol. %</td>
<td>84</td>
<td>40-50</td>
</tr>
<tr>
<td>2. R.V.</td>
<td>11.6</td>
<td>66</td>
<td>20-40</td>
</tr>
<tr>
<td>3. R.A.</td>
<td>12.16</td>
<td>49</td>
<td>0-10</td>
</tr>
<tr>
<td>4. B.A.</td>
<td>17.37</td>
<td>99</td>
<td>10-15</td>
</tr>
<tr>
<td>5. Capacity</td>
<td>17.53</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Pulmonary Flow = \( \frac{353}{173.7-147.9} \) = 353 \( \frac{25.8}{57.7} \) = 13.68 L./min.

Peripheral Flow = \( \frac{353}{173.7-116} \) = 353 \( \frac{6.11}{57.7} \) L./min.

Shunt (Left to right) = 13.68 - 6.11 = 7.57 L./min.
P.A.—Pulmonary artery.
R.V.—Right ventricle.
R.A.—Right auricle.
B.A.—Brachial artery.
Oxygen consumption = 353 cc./min.

The results are summarized in table 1. The findings revealed an elevated pulmonary artery pressure and a high oxygen saturation of blood from the pulmonary artery. These results indicated a definite left to right shunt which could be explained only on the basis of some lesion similar to a patent ductus arteriosus.

After catheterization the patient's blood pressure was 100/50, and for several hours the diastolic component of the previously described murmur was inaudible. However, the next day both systolic and diastolic murmurs were again audible, as originally described. The clinical diagnoses were changed to syphilitic aneurysm with rupture into pulmonary artery, congestive heart failure and left hydrothorax. The patient's course was progressively downhill with gradual increase in the size of the heart, though there was some diminution in edema. During this period the patient was able to be partially ambulatory, and on Nov. 18, 1949 he left the hospital against medical advice. The patient expired at his home on Nov. 25, 1949.

Postmortem Examination. On Nov. 29, 1949, an autopsy was performed by Dr. C. M. Phillips, Jr. The final anatomic diagnoses were (1) syphilitic aortitis, (2) aneurysm of aorta with rupture into pulmonary artery, (3) myocardial hypertrophy and dilatation, (4) aortic insufficiency, (5) chronic passive congestion of lungs and viscera, (6) early bron-

Fig. 2. Photograph of heart showing section through aneurysm of aorta revealing pulmonary artery.

chopneumonia, (7) chronic pericarditis. The heart (fig. 2) weighed 575 Gm. The right ventricular musculature measured 0.7 cm., and the left ventricular musculature measured 1.9 cm. Considerable hypertrophy of the right auricle and ventricle with much dilatation of the chambers was present. There was moderate hypertrophy and dilatation of the left ventricle. There was fibrosis of the aorta and margins of the aortic valves. The septal cusp of the aortic valve was rather tense and flattened against the wall of the corresponding sinus. There was a circumscribed area of sclerosis with bulging involving the anterior surface of the proximal portion of the aorta for a distance of 6 cm. and measuring 4.5 cm. in width. There was longitudinal wrinkling of the
wall and the proximal portion of this area. This area bulged some 1 cm. outside the diameter of the proximal aorta. Near the center of this bulging area and 1.5 cm. above the aortic valve there was a defect in the wall of the aorta 2 cm. in diameter (fig. 3). This defect communicated with a saccular aneurysm 7.5 cm. in diameter. The aneurysm projected anteriorly and into the left anterior pleural cavity displacing the pulmonary artery laterally to the left and posteriorly. The wall of the aneurysm varied from 0.3 to 2.0 cm. in thickness, and included a laminated blood clot on the anterior side. In the posterolateral portion of the aneurysm was a slit-like defect which measured 1.5 cm. long which communicated with the pulmonary artery 0.8 cm. above the pulmonary valve.

Microscopic examination of a section from the septum of the left ventricle, including the aortic valve and the wall of the aneurysm, revealed moderate myocardial hypertrophy with scarring at the base of the aortic valve. Dense hyalinized fibrous tissue encroached upon the myocardium with vascularization of this area. Adjacent to this area was the dense, hyalinized, partially calcified, fibrous wall of the aneurysm. A few foci of plasma cells and lymphocytes were noted in the scarred area with vascularization and focal scarring of the included aorta. Examination of a section from the ascending aorta revealed patchy fibrosis of the muscularis which extended to the inner third of some areas, and was associated with vascularization of the muscularis. Occasional foci of lymphocytic and plasma cell infiltrations of the adventitia and endarteritis obliterans of the adventitia were noted. There was hyalinization and thickening of the intima. The section from the ruptured area of the aneurysm into the pulmonary artery revealed vascularization and hyalinization of the pulmonary artery at this point with perivascular and lymphocytic and plasma cell infiltration. The wall of the aneurysm was composed of dense hyalinized fibrous tissue.

**Comment and Discussion**

The history in this case leaves some doubt as to the exact time of onset of the rupture; however, the history of sudden onset of pain in the left chest one year prior to admission might indicate that partial rupture occurred at that time. The previous history of a cough for about five years would suggest the development of symptoms referable to an aneurysm. According to the patient’s history, severe symptoms relative to failure did not begin until about five weeks prior to admission, and it is possible that the defect became larger at that time. On the other hand, the history could be interpreted to indicate that the actual rupture occurred five weeks prior to admission. With the pathologic changes of the aortic cusps, noted at autopsy, it is entirely possible that the patient had had an aortic insufficiency for several years. This feature may have contributed to the etiology of the left ventricular hypertrophy.

Fluoroscopy and x-ray films failed to reveal an aneurysm of the aorta, and this, coupled with the prominent pulmonary conus and general cardiac enlargement, favored the diagnosis of patent ductus arteriosus. However, a review of the patient’s history, added to the findings of cardiac catheterization, pointed the way to a correct diagnosis in this case. As noted in table 1, results of cardiac catheterization revealed a shunt of 7.57 liters per minute. This may not represent the true size of the shunt, since the sample was taken from the right pulmonary artery. The oxygen consumption was high but the patient was not basal. The peripheral flow was a little low for the oxygen consumption as indicated by the high arteriovenous difference. The catheterization studies revealed a marked increase of the oxygen saturation of the pulmonary arterial blood and a significant increase of the pressure within the pulmonary artery.

As pointed out by Klein and Porter, three factors may result in the elevation of pulmonary arterial pressure; these are (1) compression of the pulmonary artery by the aneurysm, (2) the obstructive “watergate” effect, and (3) increased volume of blood in the pulmonary circuit.
In 1942, Porter presented three case reports and an excellent summary of the symptoms and signs usually noted in this syndrome. He pointed out that a preponderance of right ventricular failure usually appeared soon after onset and that the signs of pulmonary stasis were usually slight when compared with the amount of dyspnea present. He noted that the cardiac enlargement was usually not of the aortic type and that the electrocardiogram may go on to right axis deviation when this is not evident at the onset. He felt that the marked dyspnea was secondary to an increased Hering-Breuer reflex which was the result of marked pulmonary engorgement and not secondary to left ventricular failure. Right ventricular strain is usually prominent in these cases and can result from compression of the pulmonary artery by the aneurysm or from increased pressure within the pulmonary artery as a result of the communication between the aneurysm and the pulmonary artery.

On Nov. 4, 1949, a repeat electrocardiogram on this patient revealed a tendency to right axis deviation and on Nov. 15, 1949, the electrocardiogram revealed definite right axis deviation. This finding is consistent with the findings noted in most of the previously reported cases. However, in the case report by Klein and Porter, a normal axis was present.

**SUMMARY AND CONCLUSIONS**

1. A patient with syphilitic aneurysm of the ascending aorta with rupture into the pulmonary artery is presented.

2. Cardiac catheterization was performed with results indicating a left to right shunt in blood flow. This finding coupled with a detailed history reveals the usefulness of cardiac catheterization as a valuable adjunctive tool in the diagnosis of the above syndrome.

**REFERENCES**


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