Table 1—Ascites with Sarcoidosis

<table>
<thead>
<tr>
<th>Case, Sex, Race, Age (Yr)</th>
<th>Race</th>
<th>Previous Sarcoid</th>
<th>PPD</th>
<th>Ascitic Fluid</th>
<th>TB Cultures</th>
<th>Histologic findings of Noncaseating Granulomas</th>
<th>Treatment and Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, F, B, 39</td>
<td>Skin; pulmonary adenopathy</td>
<td>Negative; anergic</td>
<td>6 L of turbid yellow fluid</td>
<td>None</td>
<td>Omentum; peritoneal cavity; intestine</td>
<td>No treatment; died 4 yr later of cor pulmonale; no ascites at autopsy</td>
<td></td>
</tr>
<tr>
<td>2, M, ?, 24</td>
<td>Parotid gland</td>
<td>Negative; anergic</td>
<td>4 L of yellow fluid</td>
<td>Negative</td>
<td>Peritoneum; lymph nodes; liver</td>
<td>Sodium restriction; ascites for 1 yr; controlled with prednisone</td>
<td></td>
</tr>
<tr>
<td>3, F, W, 28</td>
<td>None</td>
<td>Negative; anergic</td>
<td>20 L of serous fluid</td>
<td>Negative</td>
<td>Spleen; pancreas; lymph nodes; liver</td>
<td>Anti-TB therapy; no recurrence at 2.5 yr</td>
<td></td>
</tr>
<tr>
<td>4, F, W, 63</td>
<td>None</td>
<td>Positive for therapy; active TB 30 yr ago</td>
<td>2 L of serous fluid</td>
<td>Negative</td>
<td>Liver; peritoneum; hernia sac</td>
<td>Anti-TB therapy 30 yr ago; no recurrence at 2 yr</td>
<td></td>
</tr>
<tr>
<td>5, M, W, 67</td>
<td>None</td>
<td>Negative; positive 2 yr later</td>
<td>Unstated amount of turbid fluid</td>
<td>Negative</td>
<td>Peritoneum</td>
<td>Prednisone, 1 yr; ascites resolved; asymptomatic at 2 yr</td>
<td></td>
</tr>
<tr>
<td>6, F, B, 42</td>
<td>Parotid gland; liver pulmonary hilar lymph nodes</td>
<td>Negative</td>
<td>7 L of bloody fluid</td>
<td>Negative</td>
<td>Bone marrow; falciiform ligament; liver; fallopian tubes; ovaries; uterine serosa; hernia sac</td>
<td>Anti-TB therapy, 6 wk; persistent ascites for 8 yr; no ascites past 2 yr</td>
<td></td>
</tr>
<tr>
<td>7, F, B, 29</td>
<td>Pulmonary hilar lymph nodes; uveitis</td>
<td>Negative</td>
<td>4 L of bloody fluid</td>
<td>Negative</td>
<td>Cervical lymph nodes</td>
<td>Anti-TB therapy, 1 yr; prednisone, 0.5 yr; no recurrent ascites at 10 yr</td>
<td></td>
</tr>
</tbody>
</table>

this remains to be proven. The clinical course of sarcoid-induced ascites is surprisingly benign, and the condition resolved in five of the seven reported cases within a few weeks (Table 1). This is strikingly different from that seen in patients with ascites secondary to portal hypertension complicating hepatic sarcoidosis, where bleeding from the esophageal varices and death are common.9

In any case, sarcoidosis should be added to the list of those diseases which in their clinical course may produce peritoneal involvement and bloody ascites.

ACKNOWLEDGMENTS: Patient 1 is reported through the courtesy of Dr. D. Guerry, and patient 2 was under the care of Dr. H. Smits.

REFERENCES


Spontaneous Aneurysm of a Patent Ductus Arteriosus In an Elderly Patient*

J. Taylor Hays, M.D.†

Spontaneous aneurysm of a patent ductus arteriosus has rarely been reported in an adult. Successful operation on such an aneurysm is reported.

An aneurysm of the ductus arteriosus is a rare cause of a mediastinal mass. Only 14 cases have been reported in the English medical literature.1* The case presented here has unique features not seen in previous case reports.

CASE REPORT

A 69-year-old white man was admitted to the Nashville Veterans Administration Hospital for increasing dyspnea and fatigue. Four months prior to this hospitalization, while undergoing evaluation for

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Spontaneous Aneurysm (J. Taylor Hays)
dyspnea, pedal edema, and atrial fibrillation, a mediastinal mass at the level of the aortic knob was discovered on routine chest roentgenogram (Fig 1). A CT scan of the chest with contrast revealed a vascular mass filling the aortopulmonary window. It was believed to represent either a pulmonary artery aneurysm or dissecting aortic aneurysm. Because of his pulmonary disease, no further studies were done. He gradually deteriorated at home and was readmitted.

On examination, blood pressure of 110/50 mm Hg and regular pulse rate of 84 beats per minute were noted. He was hoarse and had marked elevation in jugular venous pressure. Cardiac examination revealed a right ventricular heave and a grade 3/6 holosystolic murmur at the left third intercostal space, as well as a short and soft diastolic murmur. There was hepatomegaly and pitting pretibial edema. He had no clubbing or cyanosis. A chest roentgenogram again revealed the mass and elevation of the left hemidiaphragm. Fluoroscopy confirmed paralysis of the left hemidiaphragm and direct laryngoscopy showed left vocal cord paralysis.

Aortography revealed an aneurysm in the aortopulmonary window which filled quickly with contrast and communicated directly with the pulmonary artery (Fig 2). Right and left heart catheterizations demonstrated mild to moderate pulmonary hypertension and a marked step-up in the oxygen saturation from right ventricle to main pulmonary artery. These data were consistent with a large left-to-right shunt at the level of the ligamentum arteriosum.

A left posterolateral thoracotomy was performed and the patient placed on cardiopulmonary bypass. A large aneurysm approximately 10 cm in its largest diameter was identified, situated between the aortic arch and the pulmonary artery. Its wall was calcified in areas and was so thin that blood could be seen flowing within the aneurysm. No clot was evident in the aneurysm. A 2.5 x 3.0 cm ostium opening into the inferior wall of the aortic arch was identified as well as a 1.5 x 2.0 cm ostium opening into the left pulmonary artery just distal to the bifurcation of the main pulmonary artery. The aneurysm was resected and the aortic and pulmonary artery defects repaired. Microscopic study of the specimen revealed intima and atherosclerotic changes with focal calcification and fibrin deposits. The patient suffered an intraoperative left cerebral hemisphere infarct but presently is doing well with only mild residual dysphasia and right arm weakness.

**Discussion**

Spontaneous aneurysm of a widely patent ductus arteriosus (DA) has never been reported in an adult. Aneurysm formation in a previously ligated but recanalized DA has been reported in both children and adults.8,9,10 According to Taussig,10 aneurysmal dilatation of the DA occurs only when obliteration of the DA is complete at the pulmonary end but incomplete at the aortic end, allowing the diverticulum thus formed and exposed to systemic arterial pressures, to dilate over time. A recently reported series from Stanford has confirmed this concept.8

The unique features of this case are formation of the aneurysm in a widely patent DA and many presenting symptoms caused by pulmonary circulatory overload from left-to-right shunting through the aneurysm. The patient developed increasing dyspnea and edema initially thought to be due to congestive heart failure, but a 64 percent global ejection fraction indicated normal left ventricular function. Most of his symptoms were caused by pulmonary hypertension secondary to chronic volume overload from the large volume of blood shunted from systemic to pulmonary circulation. As his DA aneurysm enlarged, a larger volume of blood was shunted and his symptoms worsened.

The symptoms and signs caused by the mass effect of the aneurysm, such as hoarseness from recurrent laryngeal nerve palsy and widening mediastinum on chest roentgenogram, are not uncommon presenting manifestations of this problem as previously reviewed.8 As the aneurysm gradually enlarges, the danger of spontaneous rupture or erosion into the pulmonary artery,7 major bronchi,9 or esophagus10 increases making the need for operative intervention urgent.

The diagnosis of a DA aneurysm can only be confirmed by arteriography in a patient whose presenting features suggest the diagnosis. Computed tomographic scanning with contrast enhancement can be a useful noninvasive means of determining which patients should proceed to further invasive tests. If, as in this case, the mediastinal mass is shown to be a vascular structure, then arteriography is necessary.

As in other reports, the surgical approach to this problem is complicated by the large size these aneurysms can attain and by the friability of the often-calcified tissue. Cardiopulmonary bypass is required for aneurysm resection and repair of the major vessels.8 As noted above, because of the often

**Figure 1.** Widened mediastinum (left) compared to 15 months before (right).
Marked Suppression of Ventilation While Awake following Massive Ingestion of Atenolol

A. Bruce Montgomery, M.D.; Marie A. Stager, B.S.N.; and Robert B. Schoene, M.D.

We describe a patient who ingested 5 g of atenolol with ethanol. After awakening, with repeat toxicology screen only showing atenolol, and in spite of normal voluntary breathing mechanics, he had marked suppression of his spontaneous respirations as measured by minute ventilation and by occlusion pressure with no incremental response to hypercapnic challenge. Subsequently, he recovered. Although we are unable to prove a causal relationship, future patients with atenolol overdose should be observed carefully for ventilatory failure, even if fully conscious.

Self-poisoning with large doses of propranolol, a beta-adrenergic antagonist, have been reported frequently. Physiologic effects include bradycardia, heart failure, hypertension, bronchospasm, hypoglycemia, and seizures. Only one case has been reported of self-poisoning with atenolol, a new adrenergic receptor blocking agent with greater beta selectivity. We report a patient who ingested 5 g of atenolol, four times the previously reported amount and who temporarily had marked suppression of spontaneous ventilation while awake. This drug-induced ventilatory suppression was similar to the central nervous system-mediated alveolar hypoventilation which has been given the eponym, Ondine's curse.

Case Report

A 42-year-old man had been taking atenolol for two years for treatment of mild hypertension. In a suicide attempt, he ingested

References

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