Atraumatic Right Ventricular Aneurysm

Report of Case

By William B. Weglicki, M.D., Jerome Ruskin, M.D.,
and Henry D. McIntosh, M.D.

The development of an aneurysm of the left ventricle is a relatively common sequela of myocardial infarction.1–4 The pathophysiological consequences of such aneurysms have been defined by hemodynamic and cinefluorographic studies both before and after successful resection.5–8 In contrast, aneurysms of the right ventricle are rare and coronary artery disease is not thought to be an important etiological factor. The majority of such aneurysms are secondary to trauma or surgical procedures involving the right ventricle.9–18 Comparatively few successful surgical resections of aneurysms of the right ventricle have been reported.9, 13–15, 18

The present communication describes a patient with an aneurysm of the right ventricle which, unlike that in most other reported cases, developed without antecedent surgery or trauma. Hemodynamic and cinefluorographic data were obtained at cardiac catheterization. Surgical resection of the aneurysm was accomplished.

Report of Case

H. B., a 44 year old white man, had been admitted to the Duke University Medical Center 8 years prior to the present admission. At that time he complained of fever, migratory polyarthralgias, fatigue, and loss of weight. Examination strongly suggested the presence of polyarteritis nodosa, but this diagnosis was never clearly substantiated. There was no evidence of a cardiac aneurysm in the chest x-ray at that time. Because of the possibility of a collagen disease, the patient was treated empirically with steroids and discharged. He continued to be symptomatic. Infiltrates in the right lower lung field were apparent in the chest x-ray obtained 4 months later at another hospital. There was no significant cardiac enlargement. However, in another 6 months, the chest film demonstrated enlargement which was compatible with, but not diagnostic of, pericardial effusion. Over the next 5 years the patient received intermittent steroid therapy for vague, ill-defined symptoms. During the year prior to the present admission, he developed paroxysmal supraventricular tachycardias and mild congestive failure and was treated with digitalis and quinidine. He experienced progressive fatigue and dyspnea on slight exertion; episodes of palpitations increased the dyspnea.

The development of atrial fibrillation prompted the readmission to the Duke University Medical Center in June 1965. Pertinent physical findings on admission included blood pressure of 110/70 mm Hg and an irregular pulse of 80/min. The venous pulsations in the left anterior triangle of the neck were prominent and of much greater amplitude than the pulsations of the right superficial veins; the veins of the right arm were more distended than those of the left. The lungs were clear to percussion and auscultation. The point of maximal cardiac impulse was at the midclavicular line. A grade I/VI early systolic ejection murmur was audible in the second intercostal space along the left sternal border. A faint ventricular diastolic gallop was also present.

The chest x-ray demonstrated a bulging mass in the region of the left hilum (fig. 1). Review of all previous chest x-rays indicated that the aneurysmal bulge developed between 1957 and 1961. Except for the preadmission electrocardiogram, which demonstrated atrial fibrillation, repeated tracings were normal except for occasional premature ventricular contractions and digitalis effect. At fluoroscopic examination the mass was seen to pulsate in a paradoxical manner in the area of the right ventricle. During cinefluorography contrast media injected into the right atrium

From the Cardiovascular Laboratory, Department of Medicine, Duke University Medical Center, Durham, North Carolina.

This study was supported in part by Program Project Grant HE-07563, and Grants H-4807 and HTS-5369 from the U. S. Public Health Service, National Heart Institute of the National Institutes of Health, and grants-in-aid from the John A. Hartford Foundation, The Council for Tobacco Research, U.S.A., and the North Carolina Heart Association.
(fig. 2) opacified a large dilated right atrium; the body of the right ventricle was not enlarged but appeared to be tipped upward and displaced medially from the apex. The right ventricular outflow tract was distorted by a saccular aneurysmal outpouching. The pulmonary arteries were normal. The aneurysmal sac pulsated paradoxically, distending during systole and emptying incompletely during diastole. Selective coronary arteriography revealed no gross pathologic changes. Retrograde injection of contrast media into the innominate vein through the femoral vein catheter did not opacify the right subclavian and deep jugular veins. Intracardiac electrocardiographic findings were not compatible with Ebstein's anomaly.

Open-heart surgery with total cardiopulmonary bypass was performed and successful resection of the thin-walled right ventricular aneurysm was accomplished without difficulty.* The aneurysm was observed to pulsate paradoxically during surgery, and supporting the aneurysm by hand appeared to increase the tension of the pulmonary artery. Pressure tracings of the right ventricle and pulmonary artery were obtained by direct needle puncture during surgery (table 1).

No histological evidence of abnormal myocardial tissue was observed by microscopic examination of the resected aneurysmal wall. No mural thrombi were present.

The patient had an unremarkable postoperative course. Cardiac catheterization was performed 3 weeks postoperatively, and hemodynamic data were similar to the preoperative values except for a slight decrease in cardiac output (table 1); cinefluorography showed the absence of the major portion of the aneurysm (fig. 3).

The etiology of this right ventricular aneurysm was not clear; however, its evolution and pro-

*Surgery was performed by Dr. David Sabiston, Jr., Professor and Chairman of the Department of Surgery, Duke University Medical Center, Durham, North Carolina.
Table 1

<table>
<thead>
<tr>
<th>Site</th>
<th>Preoperative</th>
<th>Blood pressure (mm Hg) Operative</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrium</td>
<td>13/5 (9)</td>
<td>—</td>
<td>14/6 (10)</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>18/4-10</td>
<td>11/4</td>
<td>16/4-12</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>—</td>
<td>11/7</td>
<td>—</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>2.7 L/min</td>
<td>—</td>
<td>2.0 L/min</td>
</tr>
</tbody>
</table>

Figure 3

Postoperative right ventricular cinefluorogram revealing the absence of the right ventricular aneurysmal sac.

gression over an 8-year period was well documented radiographically. The ventricular wall weakness was thought to be secondary to previous infection. The normal coronary arteriograms and the absence of histological evidence of muscle pathology in the aneurysmal wall militated against a diagnosis of coronary artery disease. Review of all the myocardial sections taken at the time of operation revealed no evidence of polyarteritis nodosa or infection.

Discussion

The rarity of right ventricular aneurysms in comparison to those of the left ventricle has been well documented in the literature.1–3, 9, 10 The reasons for this difference in incidence are uncertain but the frequent occurrence of myocardial infarction in the left ventricular wall and the hemodynamic differences between the greater and lesser circulations are significant factors.1–4 Aneurysms due to congenital malformations have been found in both ventricles,18–20 but they are rare in relation to the number of acquired lesions that have been diagnosed in vivo and at the autopsy table. Accidental9, 16, 17 and operative8, 10–15 trauma comprise the overwhelming majority of etiological factors leading to the development of right ventricular aneurysms.

The paradoxical movement of such aneurysms during ventricular systole is thought to lead to reduced stroke volume. Predisposition to rupture has been described, but it is not a common occurrence. The aneurysms may also serve as a nidus for thrombus formation. In our laboratory we have studied a second patient with a right ventricular aneurysm who did have pulmonary emboli which were thought to arise from the aneurysmal defect; however, no cinefluorographic evidence of thrombi in the aneurysm was noted at the time of cardiac catheterization. No further emboli have occurred in this patient since the institution of anticoagulant therapy.

In the case presented, the first suspicion of a myocardial lesion was prompted by an unusual left hilar shadow on routine chest x-rays. Unequivocal confirmation of the existence of a right ventricular aneurysm was obtained by cinefluorographic delineation of the lesion (fig. 2). Coronary arteriography was performed but did not demonstrate evidence of coronary artery disease. The absence of a positive history of previous trauma, surgery, infarction, or other pathology precluded definitive etiological classification. The age of the patient at the time the aneurysm developed seemed to militate against a congenital origin. The appearance of the aneurysm after an illness in which pneumonitis and loculated
pleural effusion were documented and pericarditis was suspected, strongly suggested that the cause was an inflammatory process. The presence of normal myocardial tissue and the absence of coronary insufficiency were compatible with this thesis.

The decision to resect the aneurysm was made because of the incapacitating symptoms of recurrent supraventricular tachycardia, fatigue, and congestive failure. Successful resection of the aneurysm was accomplished, and the patient had an uneventful postoperative course. He was restudied by cardiac catheterization (table 1), and cinefluorography (fig. 3) demonstrated the absence of the aneurysmal bulge. Unfortunately, the desired improvement in the cardiac output was not demonstrated, but the patient has improved clinically during the 5 months since discharge.

References
Atraumatic Right Ventricular Aneurysm: Report of Case
WILLIAM B. WEGLICKI, JEROME RUSKIN and HENRY D. MCINTOSH

_Circulation_. 1966;34:123-126
doi: 10.1161/01.CIR.34.1.123

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1966 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/34/1/123.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/