Post-Traumatic Aneurysm of the Left Ventricle

By Duncan A. Killen, M.D., Walter G. Gobbel, Jr., M.D., Richard France, M.D., and Vernon A. Vix, M.D.

SUMMARY

Three cases of left ventricular aneurysm secondary to external violence are reported. Each patient presented with a pseudoaneurysm of the left ventricle, following penetrating trauma in two instances and blunt trauma in the third instance. The interval between injury and diagnosis ranged from 5 months to 24 years. Two patients underwent successful resection of the aneurysm. Repair was not attempted in the third patient. Only 16 cases of post-traumatic left ventricular aneurysm have previously been reported. In 13 of the 19 cases available for review, the trauma was blunt and in six penetrating. The aneurysm could be determined to be a true aneurysm in five instances and a false aneurysm in eight instances. Complications (rupture, cardiac failure, embolism, and arrhythmia) proved fatal in eight cases. Each of the eight patients subjected to aneurysmectomy survived.

Additional Indexing Words:
Cardiac trauma Ventricular aneurysm Pseudoaneurysm of heart
Aneurysmectomy

The majority of cardiac aneurysms arise from the left ventricle and are secondary to myocardial infarction. Occasionally, left ventricular aneurysms of another etiology (such as congenital, infectious, iatrogenic) are observed. Rarely, left ventricular aneurysms secondary to external violence are observed. The observation of three patients with a post-traumatic aneurysm of the left ventricle prompted the present report.

Report of Cases

Case 1

This 44-year-old Negro man was first admitted to the Nashville Veterans Administration Hospital on July 24, 1964. He was referred because of a “lesion” on a routine chest roentgenogram. Twenty-four years prior to admission the patient had sustained a penetrating knife wound of the left chest. There had been temporary loss of consciousness. He had received only expectant treatment and was discharged from the hospital after a few days. Subsequently, he had experienced no symptoms referable to the heart or chest.

Physical examination was not remarkable. The heart sounds were normal, and there were no murmurs. The electrocardiogram revealed nonspecific S-T segment and T-wave changes. Plain chest roentgenograms revealed a mass protruding from the left border of the heart (fig. 1). Intravenous angiograms were interpreted to reveal no contrast filling of the mass. On August 11, 1964, the patient underwent exploratory thoracotomy. There was an expansive aneurysm approximately 3 cm in diameter extending from the anterolateral aspect of the left ventricle immediately to the left of the anterior descending coronary artery and just inferior to the circumflex coronary artery. The aneurysm appeared to be thick-walled. Because of the intimate relationship of the aneurysm to the coronary arteries, exploration only was performed. Postoperative periodic outpatient visits revealed the radiographic shadow to remain essentially unchanged.

The patient was readmitted in May 1968 for further diagnostic studies. There were no symptoms referable to the cardiorespiratory system. On physical examination there was a small scar over the anterior left fourth intercostal space as well as a left posterolateral thoracotomy scar.
normality (fig. 2). The phonocardiogram was normal except for either a split first sound or a faint early systolic murmur. The apexcardiogram was normal. Left ventricular cineangiograms revealed an aneurysm 3 cm in diameter, communicating with the left ventricular lumen via a channel 5 mm in diameter (fig. 3). Left ventricular pressure was 125/1 mm Hg with an end-diastolic pressure of 8 mm Hg. No operative intervention has been planned.

Case 2

This 36-year-old Negro man was admitted to the Nashville Veterans Administration Hospital for the first time on September 12, 1967, because of vague pain in a left arm amputation stump. Two years prior to admission the patient had been in an automobile accident, at which time he suffered traumatic amputation of the left arm. In addition, he suffered contusions of the chest wall (presumably from the steering wheel). There was a radiographic shadow in the right lung field thought to represent contused lung. The cardiac silhouette was normal. No electrocardiogram was obtained. The pulmonary shadow cleared, and the patient was discharged after healing of the amputated arm. During the 2 years prior to admission the patient had been plagued by phantom pain of the left arm and discomfort in the stump. During this time he had experienced no symptoms referable to the heart or chest.

Figure 2

Case 1, Electrocardiogram. Iso-electric T wave in lead I and slight elevation of S-T segment in leads V4 to V6.
Figure 3

Case 1. Left ventricular cineangiogram in right anterior position. White arrows outline lumen of aneurysm. Black arrows point to communicating canal.

Figure 4

Chest roentgenogram in case 2. Arrows outline aneurysm.

On physical examination there was a well-healed stump of the left arm. A grade II/IV ejection systolic murmur was heard over the left sternal border and at the apex. Chest radiograms revealed a mass contiguous with the heart and presenting in the anterior mediastinum (fig. 4). Old healed fractures of the left second and third ribs were also noted. Electrocardiogram was not remarkable except for low voltage of the

Figure 5

Case 2. Left ventricular cineangiogram in right anterior oblique position. White arrows outline lumen of aneurysm. Black arrows point to communicating canal.

Figure 6

Chest roentgenogram in case 3. Arrows outline aneurysm.
T-waves in lead I and the lateral precordial leads. The presence of an early systolic murmur was documented by phonocardiography. Exploration of the anterior mediastinum through a median sternotomy revealed the mass to be expansile and to be within the pericardial sac. Exploration only was performed. The postoperative course was unremarkable. Following recovery, left heart catheterization revealed a left ventricular pressure of 140/0 mm Hg with an end-diastolic pressure of 10 mm Hg. Left ventricular cineangiograms revealed the presence of a 6-by-8 cm aneurysm communicating with the left ventricular lumen via a narrow neck (fig. 5).

On November 8, 1967, the patient underwent cardiopulmonary bypass with resection of the aneurysm and suture closure of the (0.8 cm by 1 cm) communicating defect. The aneurysm wall consisted of fibrous tissue with no evidence of myocardial fibers. The patient's postoperative course was unremarkable. He was well when last evaluated 6 months postoperatively.

**Case 3**

This 39-year-old Negro man was admitted to the Nashville Veterans Administration Hospital on December 30, 1967, with a complaint of chest pain of 3 days' duration. Four months prior to admission, the patient had been admitted to another hospital after sustaining a penetrating knife wound in the left anterior fourth intercostal space near the sternal border. He was seen 30 min following the injury, at which time there was a sucking wound of the chest, and the blood pressure was 80/60 mm Hg. A left thoracostomy tube was inserted, and transfusions were administered. The central venous pressure rose to 19 cm of saline, and fluoroscopy revealed a sluggish cardiac silhouette. Percardiocentesis was performed with removal of 20 ml of blood, followed by prompt improvement of the cardiovascular status. Serial roentgenograms revealed a residual left hemotorax which slowly cleared. The serum glutamic oxalacetic transaminase was 48 units on the second and 58 units on the sixth hospital day. He was discharged on the ninth hospital day with some residual fluid in the left pleural space. There was persistent blunting of the left costophrenic angle, but the cardiac silhouette was unremarkable when the patient was last seen after that hospital dismissal on November 2, 1967.

For one month prior to admission to the Veterans Administration Hospital, the patient had noted dull aching pain in both shoulders. Three days prior to admission he noted the onset of aching, continuous pain in both shoulders which at times radiated down the left arm. The pain was aggravated in certain positions. One day prior to admission he developed mild shortness of breath.

On physical examination blood pressure was 140/90 mm Hg. A harsh (grade III/IV) systolic murmur was heard over the precordium, especially toward the axilla. Chest roentgenograms revealed a large globular extension from the left border of the cardiac silhouette (fig. 5). Fluoroscopic examination confirmed that this mass was expansile and that it moved paradoxically to the left ventricular border. The electrocardiogram revealed terminal inversion of the T waves in leads I, II, and the lateral precordial leads. The phonocardiogram confirmed the presence of a loud early systolic murmur, and the apexcardiogram revealed a notch of unknown significance at the peak of the ventricular ejection wave. Left ventricular cineangiography revealed a large (8 cm by 10 cm) aneurysm with a narrow (10 mm) neck which communicated with the left ventricular lumen near the apex (fig. 7).

On January 4, 1968, the aneurysm was resected while using cardiopulmonary bypass. The communication (0.6 cm by 1.0 cm) between the left ventricle and the aneurysm was closed with interrupted sutures. The aneurysm wall consisted of organizing thrombus and new fibrous tissue without myocardial fibers. Postoperatively, the patient demonstrated a right hemiplegia and aphasia. The aphasia has cleared partially, and the cardiac status is normal; however, the hemiplegia has persisted. He was last evaluated 3 months after operation.
### Table 1

**Reported Cases of Post-Traumatic Aneurysm of Left Ventricle**

<table>
<thead>
<tr>
<th>Case</th>
<th>Reference</th>
<th>Age*</th>
<th>Type of trauma†</th>
<th>Symptoms</th>
<th>Means of diagnosis</th>
<th>Treatment</th>
<th>Follow-up†</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Potain, 1892</td>
<td>40/M</td>
<td>Blunt (13 mo)</td>
<td>Palpititation; dyspnea on exertion</td>
<td>Autopsy</td>
<td>None</td>
<td>Death (CHF?)</td>
</tr>
<tr>
<td>2</td>
<td>Groom, 1897</td>
<td>16/M</td>
<td>Blunt (1 mo)</td>
<td>Weakness</td>
<td>Autopsy</td>
<td>None</td>
<td>Death (rupture)</td>
</tr>
<tr>
<td>3</td>
<td>French, 1912</td>
<td>3/F</td>
<td>Blunt (20 days)</td>
<td>Tachycardia</td>
<td>Autopsy</td>
<td>None</td>
<td>Death (rupture)</td>
</tr>
<tr>
<td>4</td>
<td>Vaquez, 1924</td>
<td>Adult/M</td>
<td>Blunt (several mo)</td>
<td>Congestive heart failure</td>
<td>Autopsy</td>
<td>None</td>
<td>Death (CHF)</td>
</tr>
<tr>
<td>5</td>
<td>Joachim and Mays, 1927</td>
<td>25/M</td>
<td>Blunt (13 yr)</td>
<td>Arrhythmia; congestive heart failure</td>
<td>Autopsy</td>
<td>None</td>
<td>Death (arrhythmia)</td>
</tr>
<tr>
<td>6</td>
<td>Hawkes, 1935</td>
<td>6/M</td>
<td>Blunt (3 mo)</td>
<td>Malaise</td>
<td>Autopsy</td>
<td>None</td>
<td>Death (rupture)</td>
</tr>
<tr>
<td>7</td>
<td>Hildebrandt, 1938</td>
<td>27/M</td>
<td>Blunt (18 yr)</td>
<td>Cerebral embolus</td>
<td>Autopsy</td>
<td>None</td>
<td>Death (embolus)</td>
</tr>
<tr>
<td>8</td>
<td>Crawford, 1943</td>
<td>46/±</td>
<td>Stab (1 mo)</td>
<td>Weakness</td>
<td>Physical signs</td>
<td>None</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>Pitts and Purvis, 1947</td>
<td>10/M</td>
<td>Blunt (2½ mo)</td>
<td>Weakness; dyspnea on exertion</td>
<td>Autopsy</td>
<td>None</td>
<td>Death (rupture)</td>
</tr>
<tr>
<td>10</td>
<td>Cavazzuti and Forattini, 1952</td>
<td>—</td>
<td>Blunt</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>11</td>
<td>Lyons and Perkins, 1958</td>
<td>49/F</td>
<td>Stab (21 yr)</td>
<td>Multiple arterial emboli</td>
<td>Operation</td>
<td>Excision</td>
<td>Survival</td>
</tr>
<tr>
<td>12</td>
<td>Crepaldi and Dal Palu, 1961</td>
<td>34/F</td>
<td>Stab (4 mo)</td>
<td>None</td>
<td>Operation</td>
<td>Excision</td>
<td>Survival</td>
</tr>
<tr>
<td>13</td>
<td>Jamshidi and Berry, 1965</td>
<td>13/F</td>
<td>Stab (15 mo)</td>
<td>None</td>
<td>Operation</td>
<td>Excision</td>
<td>Survival</td>
</tr>
<tr>
<td>14</td>
<td>Waldhausen and associates, 1966</td>
<td>5/M</td>
<td>Blunt (15 mo)</td>
<td>None</td>
<td>Operation</td>
<td>Excision</td>
<td>Survival</td>
</tr>
<tr>
<td>15</td>
<td>Cuendet and associates, 1966</td>
<td>21/M</td>
<td>Blunt (1 mo)</td>
<td>Tachycardia</td>
<td>Operation</td>
<td>Excision</td>
<td>Survival</td>
</tr>
<tr>
<td>16</td>
<td>Glancy and associates, 1967</td>
<td>23/M</td>
<td>Blunt (2 yr, 8 mo)</td>
<td>Multiple arterial emboli</td>
<td>Operation</td>
<td>Excision</td>
<td>Survival</td>
</tr>
<tr>
<td>17</td>
<td>Present report (case 1)</td>
<td>44/M</td>
<td>Stab (24 yr)</td>
<td>None</td>
<td>Operation</td>
<td>None</td>
<td>Survival</td>
</tr>
<tr>
<td>18</td>
<td>Present report (case 2)</td>
<td>36/M</td>
<td>Blunt (2 yr)</td>
<td>None</td>
<td>Operation</td>
<td>Excision</td>
<td>Survival</td>
</tr>
<tr>
<td>19</td>
<td>Present report (case 3)</td>
<td>39/M</td>
<td>Stab (4 mo)</td>
<td>Pain in shoulders; &quot;short of breath&quot;</td>
<td>Operation</td>
<td>Excision</td>
<td>Survival</td>
</tr>
</tbody>
</table>

*Age at time of diagnosis.
†Time prior to diagnosis in parentheses
‡Mechanism of death when due to aneurysm in parentheses. CHF = congestive heart failure.
§Late death, 10 months postoperatively, due to brain stem embolus from site of aneurysmectomy.
Table 2

Pathological Findings in Post-Traumatic Aneurysm of Left Ventricle

<table>
<thead>
<tr>
<th>Case</th>
<th>Location</th>
<th>Size</th>
<th>Size of communication</th>
<th>Histology</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Apex</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>True (?)</td>
</tr>
<tr>
<td>2</td>
<td>Posterior wall</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>True (?)</td>
</tr>
<tr>
<td>3</td>
<td>Lateral wall near A-V groove</td>
<td>One-half distance from A-V groove to apex</td>
<td>“Broad base”</td>
<td>—</td>
<td>True (?)</td>
</tr>
<tr>
<td>4</td>
<td>Conus arteriosus</td>
<td>—</td>
<td>“Laceration of myocardial fibers”</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>Anterior wall</td>
<td>5 cm</td>
<td>“Broad base”</td>
<td>—</td>
<td>True (?)</td>
</tr>
<tr>
<td>6</td>
<td>Posterior wall</td>
<td>4 cm</td>
<td>—</td>
<td>—</td>
<td>True (?)</td>
</tr>
<tr>
<td>9</td>
<td>Anterolateral wall</td>
<td>7 × 8½ cm</td>
<td>2 cm</td>
<td>Degenerated myocardium</td>
<td>True</td>
</tr>
<tr>
<td>11</td>
<td>Anterolateral wall</td>
<td>Approx 5 cm</td>
<td>“Narrow base”</td>
<td>Partially calcified hyalinized fibrous tissue</td>
<td>False</td>
</tr>
<tr>
<td>12</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>False</td>
</tr>
<tr>
<td>13</td>
<td>Anterolateral wall</td>
<td>4-5 cm</td>
<td>5 mm</td>
<td>Hyalinized fibrous tissue</td>
<td>False</td>
</tr>
<tr>
<td>14</td>
<td>From left fibrous trigone, projecting into transverse pericardial sinus</td>
<td>5-6 cm</td>
<td>2 × 3½ cm*</td>
<td>Dense fibrous tissue</td>
<td>False</td>
</tr>
<tr>
<td>15</td>
<td>Anterior wall</td>
<td>“Large as an apricot”</td>
<td>2 cm</td>
<td>Connective tissue</td>
<td>False</td>
</tr>
<tr>
<td>16</td>
<td>Apex</td>
<td>3 × 3 cm</td>
<td>3 × 3 cm</td>
<td>—</td>
<td>True (?)</td>
</tr>
<tr>
<td>17</td>
<td>Anterolateral wall</td>
<td>3 cm</td>
<td>5 mm</td>
<td>False</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>Anterolateral wall</td>
<td>6 × 8 cm</td>
<td>8-10 mm</td>
<td>Dense fibrous tissue</td>
<td>False</td>
</tr>
<tr>
<td>19</td>
<td>Anterolateral wall near apex</td>
<td>8 × 10 cm</td>
<td>10 mm</td>
<td>Organizing fibrous tissue</td>
<td>False</td>
</tr>
</tbody>
</table>

*Additional 5-mm communication between aneurysm and left atrium.

Discussion

The etiology of left ventricular aneurysm is in most instances coronary artery occlusion with myocardial infarction. Rarely, left ventricular aneurysm is the result of trauma. When iatrogenic trauma is excluded, the traumatic left ventricular aneurysm is indeed rare. Each of the three cases presented in this report appears to be a post-traumatic aneurysm of the left ventricle. The remote history of trauma in case 1 is disconcerting; however, the direct temporal relationship of appearance of the aneurysm to a traumatic episode is clearer in cases 2 and 3.

Review of the literature revealed 16 previously reported cases of left ventricular aneurysm secondary to external violence. Pertinent clinical data regarding each of these cases and the three cases included in the present report are tabulated in table 1. In 13 cases the trauma was nonpenetrating (blunt), and in the remaining six a stab wound was the etiological agent. The diagnosis was confirmed at autopsy or operation in all but one case, and in this instance the diagnosis was based on physical findings. The interval between trauma and diagnosis of the aneurysm ranged from 1 month to 24 years.

The complications of post-traumatic left ventricular aneurysm are the same as those of aneurysms due to myocardial infarction. Rupture of the aneurysm was the cause of death in four instances. Congestive heart failure developed in four cases and was the mechanism of death in two. Arterial embolism occurred in three cases, a cerebral embolism being fatal in one instance. In one case arrhythmia associated with congestive heart failure was the cause of death.
Anatomic description of the aneurysm was given, at least to some extent, in 16 cases (table 2). When the trauma had been non-penetrating (blunt) in nature, the lesion was a true aneurysm (its wall having been formed by infarcted or necrotic left ventricular wall) in five instances and a false aneurysm (its wall composed of only scar tissue) in three instances. When the trauma was a penetrating (stab) wound, a false aneurysm followed in each of five instances.

Post-traumatic true aneurysms of the left ventricle mimic true aneurysms which follow myocardial infarction and are diagnosed on the same clinical grounds, that is, a left ventricular bulge demonstrating paradoxical motion, gallop rhythms, electrocardiographic evidence of an old massive infarct, and a broad-based aneurysm by left ventriculography. Post-traumatic false aneurysms, on the other hand, differ from the usual post-infarction aneurysm and present a characteristic clinical picture as follows: (1) history of penetrating or nonpenetrating chest trauma; (2) globular extension of the left ventricle with paradoxical motion; (3) frequently, an ejection systolic murmur; (4) minimal electrocardiographic abnormalities; and (5) on ventriculography, a saccular aneurysm communicating with the ventricular lumen via a narrow neck.

The first reported operative treatment of post-traumatic left ventricular aneurysm was in 1958 by Lyons and Perkins. Each of the eight patients subjected to resection of the aneurysm has survived. One late death occurred as the result of recurrent arterial embolism 10 months postoperatively.

The lethal nature of post-traumatic aneurysms of the left ventricle and the effectiveness of operative treatment seem clear (table 1). Operative intervention seems especially applicable when the lesion is a pseudoaneurysm for in such circumstances the left ventricular muscle is damaged little, and repair is technically a simpler procedure.

References
5. Potain: 1892 (cited by Bright and Beck22).
21. Glancy, D. L., Yarnell, P., and Roberts,

50 Years Ago
Role Of The Heart In Circulatory Changes

... When we speak of heart failure we must think of this organ, not as an isolated hollow mass of muscle, but as one which is brought into relation with all parts of the body and with the changes impressed on the body from without through the central nervous system. In analysing the factors involved in these complex adaptations we shall do well to start with the powers and properties of the heart itself; but we must always remember that the extraordinary powers with which the heart muscle is endowed represent but the central fortress of the system, and under normal conditions are protected and, to a large extent, prevented from coming into play by the activities of the defending positions and outposts provided by the central nervous system and its servants. It is only when these other defences fail that the heart is called upon to display those reactions which are at once brought to light in our study of the isolated organ. But no understanding of the circulatory reactions of the body is possible unless we start first with the fundamental properties of the heart muscle itself, and then find how these are modified, protected, and controlled under the influence of the mechanisms—nervous, chemical, and mechanical—which under normal conditions play upon the heart and the blood-vessels.—ERNEST H. STARLING: On the Circulatory Changes Associated with Exercise. Lecture given at the Royal Army Medical College, October 27, 1919. In: Starling on the Heart. Facsimile reprints, with comments by C. B. Chapman and J. H. Mitchell. London, Dawsons of Pall Mall, 1965, p. 153.
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