infusion at 1.9 μg/kg/min. Cardiac catheterization performed within three hours of the onset of pain showed minimal narrowing of the left coronary artery extending into the left anterior descending artery. The narrowing was felt to be hemodynamically unimportant and intracoronary nitroglycerine was not given. At this time the angina had resolved and although the magnitude of the ST segment elevation had decreased, the ST segments remained abnormal. There were no irregularities or aneurysms in this artery or in any other part of the coronary arterial tree, and the remainder of the coronary arterial tree was normal. Hypokinesis of the apex, anterior wall, and septum was present. A challenge with intravenous ergonovine to provoke spasm in the left anterior artery was not performed because of the acute anterior wall infarction.

Serial cardiac enzymes demonstrated a characteristic pattern for infarction. The maximum serum creatinine phosphokinase was 907 U/L with a 34 percent MB fraction, total LDH was elevated and LDH-1 was greater than LDH-5. The electrocardiogram demonstrated a transmural anterior wall infarction (Fig 1).

Her recovery was uneventful. Therapy with verapamil 120 mg po every six hours was begun. Two weeks after the second infarction, she was exercised on the treadmill to 75 percent of her age-predicted maximum heart rate without developing chest pain or ST segment changes. At the time of this writing, three months after the last infarction, the verapamil has been continued and she has infrequent episodes of chest pain that are not associated with ST segment changes during continuous electrocardiographic monitoring. She has not had any major change in the frequency of migraine since starting therapy with verapamil.

**DISCUSSION**

This patient's clinical course is unique. Low dose sublingual ergotamine has not previously been reported to induce coronary artery spasm sufficiently prolonged or intense to precipitate infarction. Moreover, the occurrence of a second infarction in the identical area of the initial episode of spasm several days later has not been described previously in patients without known variant angina.

Focal ischemia from therapeutic doses of ergotamine is rare in patients who have no evidence of atherosclerotic heart disease or history of variant angina. However, this patient's history demonstrates that intense spasm and infarction can occur following relatively low dose sublingual ergotamine in patients with migraine who have no evidence of underlying hemodynamically important coronary occlusive disease or previous history of chest pain. Her first infarction probably occurred because the ergotamine uncovered a previously undetected predisposition to coronary spasm. Such a predisposition to arterial spasm should be suspected in patients with migraine since migraine can be associated with Raynaud's phenomenon and variant angina. Therapeutic doses of ergotamine were apparently enough to tip the balance in favor of coronary vasospasm in this susceptible patient.

The patient's second infarction cannot be fully explained at present. It seems unlikely that the initial dose of ergotamine directly caused the infarction, that additional ergotamine was surreptitiously ingested, or that the event was fortuitous. A more probable explanation is that the initial episode of ergotamine-induced spasm facilitated spontaneous spasm by "setting off" a tendency towards spasm in the previously affected coronary artery. Endothelial cell damage to the arterial wall from the first episode of spasm, learned facilitation of alpha mediated neutral pathways or some other mechanism may have rendered the anterior descending artery at risk for recurrent spasm. At present, it is unclear whether these two events mean that she will continue to have variant angina or whether the effect was temporary; and assuming that additional ergotamine is not ingested, she will revert to her preinfarction and spasm-free status.

Some patients with migraine have an underlying vasospastic disorder making them prone to coronary artery spasm. Vasospastic effects of ergotamine may lead to intense coronary artery spasm in some patients. Physicians should be alerted to potential cardiac vasospastic effects of sublingual, low dose ergotamine in the treatment of migraine.

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**Transient Severe Mitral Regurgitation Due to Myocardial Ischemia**

D. Scott Finelli, M.D., and Jawahar Mehta, M.D.

We report a case of severe episodic mitral insufficiency and acute left heart failure in a patient with a normal mitral valvular mechanism and an isolated lesion of the left circumflex coronary artery. These episodes, felt to be the result of spontaneous papillary muscle and regional myocardial dysfunction, responded well to therapeutic measures.

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Coronary artery disease can result in mitral regurgitation by either papillary muscle dysfunction, or rarely, in approximately 1 percent of myocardial infarctions, due to rupture of the papillary muscle or chordo-papillary junction. During acute myocardial infarction, one or both papillary muscles may become ischemic resulting in dysfunction and mitral regurgitation. Previous reports have shown worsening of pre-existing mitral regurgitation during exercise-induced angina in patients with suspected or proven ischemic heart disease. This report describes angiographic documentation of spontaneous transient mitral regurgitation leading to severe pulmonary hypertension and left heart failure in the presence of coronary artery disease.

Case Report

A 43-year-old man with no previously known heart disease presented with a six-week history of recurrent shortness of breath, orthopnea, paroxysmal nocturnal dyspnea and hemoptysis. Physical examination revealed an agitated, dyspneic man with blood pressure 130/60 mm Hg; heart rate, 100 beats per minute, and respirations, 40 per minute. Prominent inspiratory rales with expiratory wheezes were heard throughout both lung fields. Cardiac examination revealed normal heart size, a loud pulmonic closure sound, an S2 gallop, and a grade 3/6 holosystolic murmur heard best at the apex. No jugular venous distension, hepatomegaly or peripheral edema were present.

Chest x-ray examination showed bilateral pulmonary alveolar infiltrates and a normal size heart. Electrocardiogram showed ventricular rate of 100 per minute, QRS axis + 60° with low QRS voltage, and T-wave flattening in V5. An M-mode echocardiogram showed dilated left atrium, normal mitral valve and left ventricular dimensions with a hypokinetic posterior wall. A Swan-Ganz catheter was inserted revealing hemodynamic data as outlined in Table 1. A prominent V wave (70 mm Hg) in the pulmonary wedge pressure tracing was noted during episodes of clinical decompensation.

Digitalis was administered and nitroprusside infusion began at 20 μg/min with subsequent hemodynamic improvement (Table 1). The patient, however, developed dyspnea and orthopnea, a loud S2 gallop with prompt elevation of pulmonary capillary wedge pressure (PCWP) when attempts were made to discontinue treatment with nitroprusside. On left and right heart catheterization, while the patient was still on therapy with nitroprusside, PCWP was 12 mm Hg with no appreciable V wave. Left ventriculogram showed posterior hypokinesis with an intact mitral valve. When nitroprusside was stopped, the patient developed a marked increase in V wave (80 mm Hg). The left ventriculogram was repeated, now showing additional anterolateral hypokinesis and severe mitral regurgitation (Fig 1). Flattening of the ST segment in lead V5 was noted on the ECG tracing. Coronary angiography revealed isolated 90 percent occlusion of the distal left circumflex artery branch of the left dominant circulation. Infusion of nitroprusside was required for angiographic resolution of the mitral regurgitation and was continued for the next 48 hours before starting oral therapy with prazosin 2 mg q 6 hrs. The resulting hemodynamics were similar to those obtained with nitroprusside (Table 1). No enzymatic evidence of acute myocardial infarction was obtained.

Table 1—Hemodynamic Values before and during Treatment

<table>
<thead>
<tr>
<th></th>
<th>Pre-Treatment</th>
<th>Nitroprusside 20 μg/min</th>
<th>Prazosin 2 mg q 6 hrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>112</td>
<td>91</td>
<td>90</td>
</tr>
<tr>
<td>Brachial artery pressure (mm Hg)</td>
<td>130/65</td>
<td>118/62</td>
<td>116/60</td>
</tr>
<tr>
<td>Mean pulmonary capillary wedge pressure (mm Hg)</td>
<td>36</td>
<td>9</td>
<td>obtained</td>
</tr>
<tr>
<td>Pulmonary artery pressure (mm Hg)</td>
<td>68/18</td>
<td>40/10</td>
<td>38/8</td>
</tr>
<tr>
<td>V wave (mm Hg)</td>
<td>76</td>
<td>12</td>
<td>10</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>5.6</td>
<td>7.6</td>
<td>8.8</td>
</tr>
<tr>
<td>Systemic vascular resistance (dynes-sec-cm⁻¹)</td>
<td>1420</td>
<td>968</td>
<td>818</td>
</tr>
</tbody>
</table>

Holosystolic murmur persisted, but was much reduced in intensity, and symptoms of heart failure decreased on this regimen.

Discussion

This patient represents an example of transient papillary muscle dysfunction resulting in severe mitral regurgitation and left heart failure. Previous reports have demonstrated intermittent severe mitral regurgitation in patients with mild regurgitation at rest and

Figure 1. Left ventricular angiogram in left lateral projection during end-systole before (upper panel) and during mitral insufficiency (lower panel). Note filling of the left atrium in the lower panel. Aortic valve (A), mitral valve (M), left atrium (LA), left ventricular catheter (C).
without evidence of primary valvular disease.\textsuperscript{5,\textasciitilde} Brody and Criley\textsuperscript{7} suggest that an abnormal sequence of contraction resulting in poor apposition of the mitral leaflets may account for acute regurgitant flow. In the case presented here, resting posterior ventricular hypokinesis was demonstrated. During transient ischemia (ST flattening), the anterior wall became hypokinetic and the patient developed acute mitral regurgitation with concomitant dyspnea, increased intensity of murmur, and an S\textsubscript{2} gallop. A soft holosystolic murmur persisted during periods of compensated function, suggestive of posterior wall and/or postero-medial papillary muscle dysfunction. Periodic compromise of the anterior wall and papillary muscle may have accounted for the dramatic worsening of the mitral regurgitation. It is also possible that an increase in vascular resistance upon discontinuation of therapy with nitroprusside accounted for severe mitral regurgitation.

The response of the heart failure to vasodilator drugs was anticipated on the basis of previous studies which have shown that a combined decrease in both left ventricular preload and afterload results in decrease in left ventricular filling pressure (LVFP) and an increase in stroke volume.\textsuperscript{4,\textasciitilde} In mitral regurgitation specifically, hemodynamic studies have shown an increase in forward stroke volume and decrease in regurgitant volume with vasodilator therapy.\textsuperscript{6} Additionally, decreasing left ventricular volume decreases the size of the mitral orifice.

In summary, we have presented angiographic documentation of intermittent severe mitral regurgitation in a patient with single-vessel coronary artery disease. These episodes and the resulting left ventricular decompensation were adequately controlled on medical therapy consisting of an appropriate inotropic agent and vasodilator.

ACKNOWLEDGMENT: We wish to acknowledge the expert secretarial assistance of Ms. Pampliette Kinsey.

REFERENCES

Myocardial Infarction in a Young Woman with Isolated Coronary Arteritis\textsuperscript{*}

Robert A. Pick, LCDR, MC, USNR; Matthew U. Glover, LCDR, MC, USNR; and W. V. R. Vieweg, Capt, USN (Ret), FCCP

A 26-year-old black woman presented with a febrile illness and subsequently sustained an inferior myocardial infarction with chest pain, CPK-MB elevation and ECG changes. Left ventriculography revealed inferior wall hypokinesis, and coronary angiography demonstrated multiple aneurysms of the coronary arteries. Findings on visceral angiography of multiple organs was normal. Various etiologies were considered; however, her clinical course was felt to be most consistent with periarteritis nodosa and steroid therapy was instituted.

Multiple aneurysms of the small and medium-sized arteries are the diagnostic hallmarks of periarteritis nodosa (PAN). There are no other specific laboratory markers for this disease; therefore, angiography, (typically of the abdominal viscera), is employed to demonstrate evidence of arteritis.\textsuperscript{1,2} Coronary artery aneurysms are commonly described at autopsy;\textsuperscript{3,4} however, coronary arteriograms of patients with PAN have not been published previously. Reported is a patient who demonstrated a necrotizing form of isolated coronary arteritis most consistent with PAN.

CASE REPORT

A 26-year-old black woman was admitted to an outlying hospital because of fever and chest pain. There was no previous history of cardiac symptoms. She had been admitted to the same hospital five months before because of peptic ulcer disease with gastrointestinal bleeding.

On examination, the blood pressure was 140/74 mm Hg. The pulse was 80 beats/minute. Temperature was 100\degree F (37.7\degree C). Tachypnea was present. There was no jugular venous distention. The lungs were clear to auscultation. No murmur, gallop or friction rub was present. The chest x-ray film showed normal findings. The initial electrocardiogram demonstrated ST segment elevation in leads 1, 2, 3, aVF, and V\textsubscript{s}. The serum creatine kinase was 305 IU (normal 0-110 IU), and the lactate dehydrogenase was 278 IU (normal up to 200 IU). A subsequent creatine kinase determination was 178 IU with a 16 percent MB fraction. A hemogram disclosed a normochromic, normocytic anemia (hematocrit 30 percent). The erythrocyte sedimentation rate was...\textsuperscript{*From the Cardiology Branch, Department of Internal Medicine, and the Clinical Investigation Center, Naval Regional Medical Center, San Diego.}

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Myocardial Infarction in a Young Woman (Pick, Glover, Vieweg)