Cotton Fiber Intraarterial Granuloma After Cardiac Catheterization

To the Editor:

An open lung biopsy obtained during evaluation of a patient with pulmonary hypertension revealed an unusual finding. A 68-year-old man presented with atypical chest discomfort, disabling dyspnea, and hypoxemia out of proportion to his moderate obstructive pulmonary disease. Pulmonary hypertension was confirmed at cardiac catheterization. A thorough evaluation did not reveal pulmonary thromboembolic disease, obstructive lung disease, interstitial lung disease, inhalation or hypersensitivity disease, collagen vascular disease, or chronic pulmonary venous hypertension. Therefore, we were concerned with other, less common disease processes such as primary pulmonary hypertension, amyloidosis, vasculitis, veno-occlusive disease, or arterial tumor emboli. Open lung biopsy did not verify any of the above, but showed a mild granulomatous interstitial pneumonitis suggestive of hypersensitivity pneumonitis. Additionally, an intra-arterial granulomatous reaction to a birefringent material identified as cotton fibers was noted (Fig 1).

The discovery of intra-arterial cotton fiber emboli and associated granulomatous arteritis was unexpected. Previous experimental work with animal (guinea-pig) models of cotton fiber emboli describe the histopathology as "resembling the arteritis of hypersensitivity," as seen in our patient. A wide variety of materials (including cotton fibers) cause foreign body emboli when introduced into the vascular system. Although rarely reported in human subjects, cotton fiber emboli were documented following cardiac catheterization in a patient with pre-existing pulmonary hypertension. That report led to discontinuing the practice of placing cardiac catheters between layers of gamgee tissue during sterilization, which can leave cotton-rubber fibers on catheters. Cotton fibers can still come into contact with the catheter from touching cotton pads during the procedure or during passage through skin sterilized with cotton swabs. We presume that one of these mechanisms was responsible for the introduction of cotton fibers into the pulmonary artery in our patient. Recognition and elimination of these potential avenues of contact will help limit the development of cotton fiber emboli following cardiac catheterization.

REFERENCES

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Reprint requests: Dr. Canessa, Via A. Gramsci 28, 40012 Calderara (Bologna), Italy

Curtailment of Cardiac Reserve in Mitral Valve Prolapse

To the Editor:

According to the cardiomyopathic theory for the pathogenesis of mitral valve prolapse, a diminished cardiac reserve may occur in this disorder, even in the absence of significant atrioventricular regurgitation. Left ventricular dysfunction is not usually detected under baseline condition, but could be revealed under the effect of...
superimposed depressor factors or during exercise stress. Since beta-adrenoceptor blockers constitute first-choice therapy for the symptomatic control of these patients, the hypothesis that their depressant effect might mask left ventricular involvement was tested. In addition, as the intrinsic sympathomimetic activity (ISA) of beta-adrenoceptor blockers is reported to confer some protection to previously jeopardized myocardium, we looked at the effects of propranolol (without) and pindolol (with ISA) in patients with mitral valve prolapse.

The study was carried out in ten men with essential mitral valve prolapse, ranging in age from 18 to 41 years (mean 29.8). Ten age-matched normal subjects served as a control group. All patients were symptomatic with physical and echocardiographic signs of mitral valve prolapse. No significant mitral regurgitation was detected and radiographic chest examination was normal in all, but Holter monitoring always showed supraventricular or ventricular dysrhythmia to be present.

All individuals were submitted for evaluation of left ventricular function by two-dimensional echocardiography and radionuclide angiography under baseline condition and following two-week periods of administration of propranolol (120 mg daily) or pindolol (30 mg daily) in a random sequence. Heart rate and blood pressure were also measured in each experimental session. Thirteen echocardiographic variables derived from radionuclide angiography were analyzed. The values of each variable under the three conditions studied were compared by nonparametric analysis of variance by the Friedman and Wilcoxon tests.

Six of the ten patients reported improvement or disappearance of symptoms with pindolol, and four also obtained symptomatic relief with propranolol.

All echocardiographic and radionuclide parameters were within normal limits in baseline condition for the mitral valve prolapse patients, and remained so during treatment with both beta-adrenoceptor blockers. However, administration of propranolol induced significant (p<0.05) decreases in mean heart rate, peak velocity of left ventricular ejection and left ventricular ejection fraction, as evaluated by RNA (Table 1); in addition, a mild hypokinesis of the apical region was detected during propranolol therapy, by qualitative assessment of the nuclear angiograms. No significant changes in blood pressure were induced by either of the beta-blockers.

While the present findings do not lend support to the cardiomyopathy hypothesis of primary mitral valve prolapse, it is possible that beta-blockade with intrinsic sympathomimetic activity may be a preferable therapeutic option for patients with such condition. However, this conclusion must be checked by longterm follow-up studies of patients to see if statistical significance will turn out into medical relevance.

Table 1—Radionuclide Angiography in Mitral Valve Prolapse (n=10)

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Propranolol</th>
<th>Pindolol</th>
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<tbody>
<tr>
<td>LVEF</td>
<td>0.67±0.08</td>
<td>0.59±0.07*</td>
<td>0.66±0.07</td>
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<tr>
<td>LVPER (s^-1)</td>
<td>2.44±0.36</td>
<td>2.00±0.46*</td>
<td>2.67±0.69</td>
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<tr>
<td>HR (b/min)</td>
<td>70±14</td>
<td>59±12*</td>
<td>68±13</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>95±9</td>
<td>92±7</td>
<td>92±11</td>
</tr>
<tr>
<td>Segmental dyskinesis</td>
<td>—</td>
<td>2 pts (apical)</td>
<td>—</td>
</tr>
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</table>

LVEF = left ventricular ejection fraction; LVPER = left ventricular peak ejection rate; HR = heart rate; SAP = mean systemic arterial pressure. *p<0.05 vs baseline and pindolol.

Emphysema vs Asthma with Antitrypsin Deficiency

To the Editor:

With the advent of replacement therapy for alpha1-antitrypsin (AAT) deficiency, certain criteria have been devised for choosing suitable patients. One criterion states that the subject should already have demonstrable pulmonary emphysema of a moderate degree (ie, FEV1, between 35 and 80 percent of predicted) so that patients with endstage disease, or those without lung disease, will not be subjected to this expensive therapy.

We have encountered an instance in which a 38-year-old man with PiZZ antitrypsin deficiency was being considered for replacement therapy. The patient had asthmatic symptoms that eventually were controlled through the use of corticosteroid therapy. Chest roentgenographic examination reveal only mild hyperinflation. Pulmonary function tests over a eight-year period show variable degrees of airway obstruction (FVC 1.28 to 2.90 L, 1.28 to 3.29 L after bronchodilator therapy; FEV1, 0.71 to 1.52 L, 0.79 to 1.54 L after bronchodilator therapy), but always with a normal diffusion capacity (Dco) performed by the single breath holding method (Dco) 76 to 102 percent of predicted; alveolar volume 69 to 97 percent of predicted; Hgb 15.3 to 17.3 gm%). Arterial blood gas levels were within normal limits, even during exercise on a treadmill.

Figure 1. CT scan of lungs showing discrete blebs consistent with early emphysema.