Editorial Comment

Right Ventricular Hypertrophy: A New "Risk Factor" for Right Ventricular Infarction?*

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The right ventricle is a somewhat strange organ: it seems to function as a pump quite well even when its wall is deliberately destroyed by a cautery or when its blood supply is severely compromised. Infarction of the right ventricle was long considered rare, on the assumption that the low pressure system had a low oxygen requirement. In recent years, following the report of Cohn et al. (1), it has become clear that acute infarction of the inferior wall is distinguished from anterior infarction by several factors, including a concomitant infarction of the right ventricle in about 30% of cases.

This complication is suspected clinically by observing distension of the neck veins, sometimes hypotension, and prominent though transient R waves in the right precordial leads of the electrocardiogram, especially lead V4R. Bedside catheterization shows right atrial pressure to be elevated (10 mm Hg) and ≥80% of the left heart filling pressure. Sometimes a fluid challenge is required to elicit the hemodynamic characteristics. Hypotension is often a result of inadequate filling of the left heart by the infarcted, dysfunctional right ventricle. Cardiogenic shock in patients with this condition has been corrected by vigorous fluid replacement. More recently, noninvasive techniques have been used to diagnose this complication. Nuclear perfusion scans or bloodpool studies with wall motion analysis are reasonably sensitive and specific. In addition, echocardiography has been used at bedside to diagnose right ventricular infarction.

The mechanism for involvement of the right ventricle is not entirely clear in some cases in inferior infarction. The obvious role of coronary anatomic distribution and pathologic obstruction was discounted by the autopsy studies of Isner and Roberts (2) and Horan et al. (3), which showed comparable degrees and distribution of disease in patients with and without right ventricular infarction complicating inferior infarction. Quantitative postmortem or angiographic measurements were not done to confirm these findings.

Role of right ventricular hypertrophy. The occurrence of right ventricular hypertrophy in right ventricular infarction was also discounted by Isner and Roberts (2) and Horan et al. (3). These investigators noted a high incidence of right ventricular dilation, which was highly correlated with increased mass at autopsy; however, because 10 of their 12 cases had healed infarction, the dilation could have been secondary to the infarction rather than a primary pathogenetic factor. Even at autopsy, the correlation of right ventricular thickness with total right ventricular mass is poor, so the data are difficult to interpret. Nevertheless, in this issue of the Journal, Forman et al. (4) adduce evidence that right ventricular infarction as a complication of inferior infarction occurs primarily in patients with right ventricular hypertrophy as defined by M-mode echocardiography. This technique is the only current method available for measurement of right ventricular thickness in vivo, but requires high resolution to minimize large percentage errors in thickness measurements spanning only a few millimeters. Previous studies have shown a good correlation of these echocardiographic measurements with autopsy data.

Forman et al. (4) showed that the right ventricular hypertrophy was associated, in this unselected series of patients, with evidence of mild but definite chronic obstructive lung disease by pulmonary function testing, even though the patients were not clinically considered to have severe lung disease. (The tests were performed an average of 19 days after infarction when possible effects of acute left ventricular dysfunction had probably subsided.) The presumption is that transient periods of hypoxemia may trigger increases in pulmonary resistance, resulting in increases in wall thickness. Because such transient episodes would be unlikely to induce marked hypertrophy, it is important to note that the right ventricle thickness was, in absolute terms, only in the upper range of normal.

Although the normal right ventricle of the dog tolerates ischemia well, the hypertrophied right ventricle does not (5), just as the hypertrophied left ventricle is more susceptible to ischemia than is the normal left ventricle. Also, the right ventricle, being a volume pump, poorly tolerates a pressure load. In the setting of acute infarction with ischemia of the right ventricle and an increased pressure load secondary to left ventricular dysfunction, a mildly hypertrophied right ventricle succumbs and becomes infarcted, whereas a normally thick right ventricle survives. The data of Forman et al. therefore complete a pathogenetic chain of events previously unlinked. Given the conflicting data on how to define and measure right ventricular hypertrophy, their data...
need to be confirmed, but an important insight has been gained.

**Clinical implications.** The clinical spin-off from these data is that one must be particularly aware of the possibility of right ventricular infarction when a patient with inferior infarction also has a history of obstructive lung disease, albeit mild, or even a history of smoking. Diagnosis by Swan-Ganz catheterization may then be indicated more aggressively than might otherwise be the case, especially because jugular venous distension is less accurate as a sign of right ventricular infarction. Volume loading or afterload reduction may then be pursued as needed. Nuclear scanning is feasible and appears to be better than echocardiography when a noninvasive technique is chosen for diagnosis. The increased complication rate of these large infarcts warrants complete evaluation both during the acute illness and during convalescence.

References


