Cardiac Rupture and Ventricular Septal Defect in Isolated Right Coronary Artery Disease

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A patient is described who had both ventricular septal defect and then cardiac rupture with death within 24 hours of the onset of acute posterior inferior myocardial infarction. At autopsy he was found to have single-vessel disease involving the right coronary artery. Isolated disease of the right coronary artery can produce unexpected, fatal mechanical complications.

In emphysema/chronic bronchitis:

Life is a matter of...
Ventricular septal rupture complicating acute myocardial infarction usually occurs in patients with anteroseptal or combined anterior and inferior infarction and is unusual in patients with single-vessel coronary artery disease. The combination of cardiac rupture and ventricular septal defect is most unusual, even though both are related to the same pathogenetic factors, and to our knowledge has not been reported in a patient with isolated obstruction of the right coronary artery. Right coronary artery disease, then, usually a relatively benign condition with a good long-term prognosis, can produce severe myocardial necrosis and fatal ventricular rupture.

The factors predisposing to the disastrous clinical course in our patient are uncertain. Prolonged and recurrent chest pain indicated that myocardial ischemia was severe, and presumably the development of a collateral blood supply was inadequate. Treatment with hydrocortisone has been implicated in impairment of healing of myocardial infarction, but seems unlikely to have been a factor in our patient, since septal rupture occurred before administration of steroids and the interval between administration of steroids and cardiac rupture was short (less than four hours). Intra-aortic balloon counterpulsation (IABC) should have protected the left ventricle by reducing preload and afterload. IABC did improve forward blood flow and stabilized the patient’s clinical condition, but the improvement was temporary. The pacemaker catheter was not involved in the cardiac rupture, since it was positioned in the right ventricle after the diagnosis of VSD had been made; cardiac rupture involved the left ventricle. Closed chest resuscitation was also probably not implicated in the genesis of ventricular rupture. Closed chest resuscitation was used terminally, after the clinical picture of tamponade had occurred.

Both the VSD and then cardiac rupture with tamponade occurred within 24 hours of the onset of symptoms. These complications usually occur several days after the onset of acute myocardial infarction. The clinical history in our patient gave no evidence that infarction had begun before the day of admission, and we have no explanation for the early appearance of ventricular rupture. The clinical course of coronary artery disease is certainly unpredictable in some patients.

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