Chronic Effusive Pericarditis Associated with Healed Myocardial Infarction

Report of a Case

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Massive effusive pericarditis is an infrequent complication of a healed myocardial infarction. The following case occurred one year after an acute myocardial infarction.

CASE REPORT

This 57-year-old office clerk was admitted to the hospital because of massive ascites and peripheral edema. He had hypertension and diabetes for ten years and angina pectoris was conspicuous for five years. He sustained an acute myocardial infarction one year prior to admission. For six months, he experienced marked dyspnea, fatigue, abdominal distention and peripheral edema.

He was severely ill, disoriented and confused. Systolic pressure was 100 mm.Hg obtained only by palpation and the pulse was regular at 120 per minute. Funduscopic examination showed arteriolar narrowing. Jugular venous pulses were distended even in the sitting position and a sharp X and Y descent was noted. Carotid pulsations were decreased. There was a wide area of cardiac dullness extending to the right of the sternum and also to the left mid-axillary line. The heart sounds were distant (Fig. 1a) and there was no murmur or gallop. There was a large left pleural effusion with fine moist rales at both bases. Ascites and marked peripheral pitting edema were prominent.

Laboratory studies were normal except for mild hyperglycemia and glycosuria. Six months after the acute myocardial infarction, the roentgenologic examination demonstrated marked enlargement of the cardiac silhouette suggestive of massive pericardial effusion. Pulmonary congestion was conspicuously absent. Serial chest x-ray studies demonstrated persistent massive pericardial effusion not responding to intensive diuretic therapy.

Serial electrocardiograms were unchanged showing sinus tachycardia and low voltage in the standard leads. Q waves were also noted in leads I, II, AVL, and a QS pattern was prominent in V1 through V6 (Fig. 3). These electrocardiographic changes were consistent with an old anterior and lateral myocardial infarction.

Severe congestive failure related to ischemic heart disease and complicated by ventricular aneurysm was the initial diagnostic impression. However, it soon became apparent that the venous engorgement, the reduced cardiac output and the poor response to anticongestive therapy were attributed to massive pericardial effusion with mild constriction. Pericardiocentesis was performed and 2000 ml. of brownish fluid was removed. Microscopic examination of this effusion showed creased red blood cells. The specific gravity was 1.008 with protein of 1 gram per cent. Results of cytologic, viral and bacteriologic studies of this transudate were negative.

Seventy-five ml. of air was injected into the pericardial cavity following pericardiocentesis and the chest x-ray film showed marked pericardial thickening with massive effusion (Fig. 4). The heart was not enlarged. He improved immediately, but the effusion reaccumulated within 48 hours. He required three additional pericardiocenteses. A pericardietomy was successfully performed ten days after admission. The thick pericardium and a massive effusion was removed. The heart was normal in size, although a large scar was present over the anterior surface of the left ventricle. The result of the histologic examination was consistent with a nonspecific pericarditis. He improved dramatically. His confusion and disorientation disappeared. Neck veins remained flat and peripheral pulses were strong. The first and second heart sounds were louder (Fig. 1b). The area of cardiac dullness was markedly reduced (Fig. 2b). Abdominal distention and peripheral edema disappeared. He remained well and returned to work in eight weeks.

The etiology of pericarditis may be viral, pyogenic, tuberculous or parasitic. Uremia, trauma, collagen disease, myxedema and neoplasm are also associated with pericarditis. Specific cases of chylopericardium and cholesterol pericarditis are also added to this classification.1,2

Pericarditis with minimal effusion is a frequent association with a transmural

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myocardial infarction. The signs of pericarditis occur a few days after myocardial infarction. The pericardial effusion is minimal except when gross hemorrhage occurs into the pericardial sac.

A later and uncommon complication of myocardial infarction is the post-myocardial infarction syndrome. Dressler's de-
scribed these patients developing chest pain two to 11 weeks following a myocardial infarction. The pericardial pain may persist for a few days to months and is associated with fever, pleuritis and pneumonitis. Pleural and pericardial effusion may be a feature, but the volume of pericardial fluid is often small not interfering with myocardial function. Our patient may have had a subclinical form of the post-myocardial infarction syndrome. This was not recognized until the clinical manifestations of massive pericardial effusion and mild constriction were conspicuous.

The etiology of this syndrome is unknown although autoantibodies have been described. In these reports, the patients with this syndrome demonstrated circulating autoantibodies. Theoretically, the necrotic myocardium acts as an antigen which is responsible for autoantibodies. The antigen-antibody reaction may result in pericarditis with effusion.

Myocardial infarction complicated by chronic effusive pericarditis has already been reported. Doyle and Grace and Nicholson reported patients with myocardial infarction associated with massive pericardial effusion of unknown etiology. Chronic idiopathic effusive pericarditis was also reported by Bedford. In his patients, however, a variety of underlying entities including al-

![Figure 3: The electrocardiogram demonstrated Q waves in leads I, II, AVL and a QS pattern in leads V1 through V6.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21428/ on 04/19/2017)
coholism, hypertension and collagen disease were described, but none had ischemic heart disease.

Regardless of etiology, the clinical features of chronic effusive pericarditis are similar. The massive pericardial effusion can be present in patients for long periods of time as evidenced by our case. Patients with this condition may remain quite active and asymptomatic. It is apparent that when fluid develops slowly, the pericardium is stretched without causing cardiac constriction. In contrast, the rapid accumulation of pericardial fluid as in hemorrhage increases the pericardial pressure abruptly causing symptoms and signs of cardiac tamponade. In our patient, the pericardial thickening and effusion was the result of a slow process not associated with constrictive symptoms and signs until later in the course of his disease.

The clinical diagnosis of massive pericardial effusion was suggested by the observation of cardiac silhouette enlargement which was disproportionate to the signs and symptoms of congestive failure. The heart sounds were soft and gallop rhythm was absent. Electrocardiographic features also showed low voltage. The x-ray film demonstrated an enlarged cardiac silhouette with only mild pulmonary congestion.10

Trendelenburg position may also shift the pericardial effusion and a change in the cardiac silhouette may be observed. Roentgenologic techniques demonstrating the actual heart border are excellent and reliable methods for differentiating massive pericardial effusion from cardiomegaly. The demonstration of the heart chambers by the injection of carbon dioxide intravenously has been helpful in differentiating cardiac enlargement from pericardial effusion. The border of the heart may also be demonstrated with the cardiac catheter and intracardiac angiocardiography may demonstrate the size of the cardiac chamber.11,12

Intravenous 131 albumin may determine the volume of blood in the heart chambers by precordial counting. The intracavitary blood volume is compared with the cardiac silhouette to determine a pericardial effusion. Pericardiocentesis is a definite method for diagnosing and evaluating pericardial effusion.4 This was done in our patient. The pericardial space may be further evaluated by injecting a small amount of air following the removal of fluid and repeating the chest x-ray film (Fig. 4). In our patient, pericardiocentesis was not only diagnostic, but also therapeutic. The results of the viral, bacteriologic and cyto-

**Figure 4:** Seventy-five ml. of air was injected into the pericardial cavity. The pericardium was thick, a massive pericardial effusion was conspicuous and the heart was not enlarged.
logic studies were negative and since gross blood was absent in the pericardial cavity, rupture of a ventricular aneurysm was not considered.

Pericardectomy was indicated because of the rapid accumulation of the massive effusion and beginning cardiac constriction. \textsuperscript{1,2,3} This promptly relieved his symptoms and signs.

References

JOINT COMMITTEE STATEMENT
TUBERCULIN SKIN TESTING OF CHILDREN

A Joint Committee of the Section on Diseases of the Chest, American Academy of Pediatrics and the American College of Chest Physicians.

Infection with tuberculosis in children is still a problem. Since early treatment with INH has been found valuable for tuberculin converters, particularly in the very young, it is mandatory that tuberculin testing be carried out early in life. This Committee strongly suggests that routine tuberculin testing be initiated between 6-12 months of age, preferable before measles or smallpox vaccination, and be repeated annually up to 4 years of age and thereafter every 2 years, depending on the risk of exposure of the child and the prevalence of tuberculosis in the population. To this end the following recommendations are made:

1. The Mantoux Test is the most accurate measure of tuberculin sensitivity. Intermediate P.P.D. (5 T.U.) is the standard test dose. The reaction to the Mantoux is read on the second or third day. The diameter or induration should be measured and recorded. Ten (10) mms. or more of induration indicates a positive reaction and 5-9 mms. induration is considered a doubtful test.

2. For clinical screening a multiple puncture test may be employed (e.g. Tine, Head). Any doubtful reaction should be checked by Mantoux testing.

3. The use of tuberculin patch tests is not recommended.

4. P.P.D. when diluted in quantities of 50 doses or more may be stored for periods of up to 6 months without significant loss of potency provided the material is kept refrigerated, sterile and away from sunlight.

PULMONARY ACTINOMYCOsis

A case of pseudotumoral form of pulmonary actinomycosis with massive invasion of the thoracic wall is reported. Left lower lobectomy and block resection of part of the thoracic wall and diaphragm were successfully carried out, with the possible chance of an enlarged radical resection for carcinoma of the lung. The pathologic study of the specimen defined the lesion as an actinomycosis of the lung invading the intercostal spaces and rib. Diagnosis and surgical techniques in either carcinoma or actinomycosis are discussed in reference to the case reported. The rarity of similar cases is emphasized in these cases.