Subacute Bacterial Endocarditis Arising in Mural Thrombi Following a Myocardial Infarction: A Case Report

By Seymour Joffe, M.D. and Harold Feil, M.D.

A case of subacute bacterial endocarditis arising in a thrombus covering myocardial infarction, with a necropsy, is reported. The patient died after an illness of four months duration. The clinical course was characterized by a low fever, congestive failure, petechiae, purpura, and ulceration of the skin. The rarity of this clinical picture is emphasized.

FEVER in a patient with heart disease frequently indicates infection or infarction of either the heart or lung. However, the occurrence of infection and infarction concomitantly in the heart is, at least statistically, extremely uncommon. Only four cases have been reported in the literature of bacterial invasion of an acute myocardial infarction followed by abscess formation in the myocardium. Similarly, a purulent pericarditis following an acute infarction on the basis of coronary artery disease is extremely rare. In a series of 77 cases of purulent pericarditis only one case of associated coronary thrombosis is recorded. In Bean’s series of 287 cases of myocardial infarction (all but five having coronary artery disease) 101 (32 per cent) had evidence of pericarditis and two of these (0.7 per cent) had a purulent process. These figures are in marked contrast to the high incidence of pulmonary infection as a terminal event in patients with coronary artery disease.

The case we are now presenting had a recent coronary artery occlusion with myocardial infarction. During convalescence he had jaundice, petechiae, purpura, and myocardial failure. This report is made because of the rarity of this clinical picture.

CASE REPORT

A 54 year old male was admitted to University Hospitals because of petechial eruption of five weeks duration. Eleven weeks prior to admission the patient had an acute myocardial infarction which was treated at home with bed rest, digitalis, and Aureomycin; the Aureomycin was given shortly after the episode of chest pain because of a suspected pneumonia. Convalescence was uneventful until nine weeks prior to admission when pretilial edema was noted. Diuretics (ammonium chloride and Mercuhydrin) were given with a fairly good response. He, however, reaccumulated edema fluid which became refractory to routine diuretic therapy. Six weeks prior to admission petechiae appeared in the lower limbs which progressed to areas of ulceration. Two weeks prior to admission the patient was hospitalized in another hospital where evidence was found of renal and hepatic dysfunction but no disturbance in the clotting mechanism. Laboratory data revealed the following: Hemoglobin 14 Gm. per 100 cc.; white blood cell count, 6500; 4 plus albuminuria with microscopic hema
turia; blood urea nitrogen 53 mg. per 100 cc.; urea clearance, 40 per cent; cholesterol 86 mg. per 100 cc. and a bromsulphalein retention of 42 per cent. Bleeding time, clotting time, clot retraction and platelet count were all normal.

When seen at University Hospitals the patient appeared acutely and chronically ill. The blood pressure was 112/100, pulse, 82 and regular in rhythm, respirations 24 per minute and temperature 37.2 C. The sclerae were icteric and there was moderately intense jaundice. There were numerous petechial and purpuric areas over the legs and arms. On the posterior aspects of both legs these areas had become ulcerated. Petechiae were also present in the buccal mucosa. The lungs were clear to auscultation and percussion. The heart was enlarged to 1 fingerbreadth beyond the midclavicular line. The heart sounds were distant. The liver extended 3 fingerbreadths below the right costal margin and the edge of the spleen was palpated on inspiration. There was 3 plus pretilial and 1 plus presacral edema. Neurological examination revealed no abnormalities.
Laboratory data found on admission were as follows: Hemoglobin 16.8 Gm. per 100 cc.; red blood cell count 5.95 million; hematocrit 65 per cent; white blood cell count 11,050, with a normal differential; platelets 252,950; bleeding time 2 min. 40 sec.; clotting time 5 minutes; clot retraction beginning in 3 hours; prothrombin time 25.5 seconds (control 34 seconds); thrombin time 34 seconds (control 30 seconds). Examination of the urine showed a specific gravity of 1.012, 2 plus albumin 50 to 60 red blood cells, and 5 to 8 white blood cells, per high power field. Total serum protein was 7 Gm., albumin 3.6 Gm., globulin 3.4 Gm.; blood urea nitrogen 30 mg. per 100 cc. The icteric index was 36; cephalin flocculation 1 plus; thymol turbidity 0.2 units; total bilirubin 7.3 mg., indirect 2.6, direct 4.7; alkaline phosphatase 8.3 units; urine urobilinogen 0.8 Ehrlich units. Sodium ranged from 120 to 128 mEq., potassium from 5.6 to 6.1 mEq., and chloride from 92 to 98 mEq per liter. Kline exclusion test was negative. Stool was positive for occult blood on three occasions. Rumpel-Leede's test was weakly positive. No bromide or barbiturate was detected in blood. The electrocardiogram showed first degree A-V block, incomplete right bundle branch block and evidence of anterior wall myocardial infarction. Culture of petechiae showed no growth, and biopsy demonstrated focal acute inflammation in skeletal muscle and subcutaneous tissue. The venous pressure was 285 mm. of saline.

On admission drug toxicity was suspected. With this in mind, drugs were given to the patient with extreme caution. Mercurials were avoided for this reason and the congestive failure increased. New showers of petechiae developed, the jaundice deepened, and bilateral pleural effusions appeared. Thoracenteses were performed on four occasions. The pleural fluid had a specific gravity ranging from 1.012 to 1.016. Large numbers of white cells (6050 to 54,900) with a preponderance of polys were found. Streptococcus viridans, Staphylococcus aureus and Aerobacter aerogenes were found. Cell blocks of the fluid were all negative for tumor cells. Bone marrow showed leukopagocytosis as sometimes seen in subacute bacterial endocarditis. Culture of the bone marrow revealed no growth. Six blood cultures showed no growth in seven days. A urine culture (yielded Proteus vulgaris and Streptococcus viridans. The patient had a low grade fever, never going above 38.2 C. His clinical condition progressively deteriorated, being marked by episodes of lethargy and confusion. On the day prior to death the patient was given test doses of penicillin and Mercuhydrin without ill effect, before planned therapy.

Postmortem examination. The skin was jaun-

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Fig. 1. Section of gastrocnemius muscle showing acute focal myositis.
**FIG. 2.** Recanalized thrombus in the anterior descending ramus of the left coronary artery.

**FIG. 3.** Showing mural thrombus in left ventricle adjacent to myocardial infarction.

**FIG. 4.** Mural thrombus in right atrium adjacent to atrial infarction.

**FIG. 5.** Section of left ventricle showing mural thrombus with bacterial invasion. Arrow points to areas with bacterial invasion, identified under higher magnification.
bacterial invasion was demonstrated on histological examination. This consisted of clumps and clusters of Gram positive cocci which were unfortunately not cultured because infection was not suspected on gross examination. The aortic valve showed subcommissural adhesions between the right and left anterior cusps. Each cusp was shortened with a thickened firm nodular margin which was probably the result of old rheumatic valvulitis. There were no vegetations.

The pleural spaces showed bilateral serous effusions (600 cc. each), and extensive pleural adhesions, many of which enclosed small pockets of purulent material. There were organizing abscesses in the right upper, right lower, and left lower lobes of the lungs with focal organizing pneumonia, pulmonary hemorrhage, and necrosis. In the left lower lobe abscesses *Aerobacter aerogenes*, *Alkaligenes fecalis*, and *Staphylococcus aureus*, (coagulase negative) were recovered. There were bilateral pulmonary infarcts with an organizing thrombus occluding the pulmonary artery to the right upper lobe (fig. 6). Microscopic section of this thrombus showed the presence of bacteria.

The esophagus was the site of an acute inflammation. The kidneys showed tubular bile casts and tubular degeneration. There was generalized hyperemia of the viscera and the remainder of the necropsy was not remarkable.

**Fig. 6.** Section of pulmonary artery with thrombus

**Discussion**

The patient died four months after myocardial infarction with a final illness characterized by congestive failure, petechiae, purpura, ulceration of the skin, and subsequently, by hematuria, jaundice, evidence of septic pulmonary infarcts and inflammatory pleural effusions. Bacteria were demonstrated in sections from mural thrombi of the left ventricle and the right atrium. We believe that infection of mural thrombi associated with myocardial infarction took place. Peripheral and pulmonary emboli resulted. The original source of infection was never demonstrated. All blood cultures were negative, as found in a small percentage of cases of subacute bacterial endocarditis.

Subacute bacterial endocarditis classically arises on a valve deformed by rheumatic heart disease, on congenital lesions, or rarely on calcareous or syphilitic valvular lesions. The mural endocardium may become secondarily involved by direct contact or extension. In this patient, while the mural endocardium was involved, the valves remained free of infection. This is extremely uncommon in either acute or subacute bacterial endocarditis. Schulz has reported a case of puerperal sepsis with a circumscribed area of mural endocardium of the left ventricle, covered with fibrin and soft thrombi while the valvular endocardium was not involved. This may also occasionally occur in pneumococcal endocarditis. Morrison cited one case of subacute bacterial endocarditis, arising only on the mural endocardium. Endocarditis occurring subsequent to a coronary occlusion is not unheard of. Libman and Friedberg reported a patient who had suffered two attacks of coronary occlusion and in whom subacute bacterial endocarditis occurred on the basis of an old aortic insufficiency. Lemann has reported a case in which the infection developed on a mitral valve which had been made insufficient because of infarction of the left posterior papillary muscle due to a coronary artery thrombosis. Libman and Friedberg noted two other cases in the literature in which a similar mechanism was involved. The unusual feature in our case is that
mural thrombi occurring secondary to myocardial infarction predisposed to subacute bacterial endocarditis. The aortic valve was the site of a healed rheumatic endocarditis and was not involved in the process.

**Summary**

A case report is presented of a patient with recent myocardial infarction with mural thrombi which became infected and led to peripheral and pulmonary embolization. The low incidence of the concomitant occurrence of cardiac infection and myocardial infarction is discussed.

**Summario in Interlingua**

Es reportate un caso de recente infarcimento myocardiac con thrombos parietal que deveniva inficite e causava embolisation peripheric e pulmonar. Es discutite le infrequentia del co-occurrentia de infection cardiac e infarcimento myocardiac.
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