Acute Respiratory Failure with Massive Pulmonary Infiltrates Six Weeks after Coronary Artery Bypass Operation

Haim Mayan MD[1], Rami Kantor MD[1], Arie Wollner MD[2] and Zvi Farfel MD[1]

[1]Department of Medicine E and [2]Division of Pulmonary Medicine, Sheba Medical Center, Tel-Hashomer,and Sackler Faculty of Medicine, Tel Aviv University, Israel

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Post-pericardiotomy syndrome is an immune phenomenon that occurs within a few months after surgical incision of the pericardium. The main clinical features are fever, pleuritis, pericarditis and, more rarely, pulmonary infiltrates. Acute respiratory distress syndrome rarely occurs as an acute complication of coronary artery bypass operation hours to several days after the procedure. We present a case of a combination of PPS and ARDS occurring 6 weeks after CABG, which was amenable to corticosteroid therapy.

Case Description

A 47 year old man with ischemic heart disease presented with acute infero-posterior myocardial infarction. Echocardiography showed an apical aneurysm and ejection fraction of 35%. A few months later he underwent CABG and was discharged with high dose steroid treatment for a quinolone-induced allergic skin reaction. Two weeks after discharge he was admitted for *Salmonella* gastroenteritis, that resolved within a few days. Six weeks after the operation, while still taking 10 mg prednisone a day, he was admitted because of high fever (39°C) and intermittent cough. Physical examination revealed pulmonary rales and a pericardial friction rub. Electrocardiography was normal. Chest X-rays showed an enlarged cardiac silhouette, a small left pleural effusion and a right lower lobe infiltrate. Echocardiogram demonstrated an enlarged left ventricle, decreased left ventricular ejection fraction (35%) and a minimal posterior pericardial effusion, all considered normal findings and not different from those before surgery, except for the minimal pericardial effusion. Laboratory investigations revealed: erythrocyte sedimentation rate 75/105, hemoglobin 10.8 g/dl, platelets 200,000/µl, white blood cells 6,800/µl with 81% polymorphonuclears and 10% lymphocytes. Blood chem-istry including creatine phosphokinase was normal except for lactate dehydrogenase 450 u/L (normal < 260 u/L). Six blood cultures were negative. Serology for *Legionella*, Q fever, *Mycoplasma, Chlamydia*, cytomegalovirus, herpes simplex, varicella zoster, enteroviruses and HIV were negative. Antinuclear factor and antineutrophil cytoplasmic antibodies were negative, complement levels were normal and the latex agglutination test was positive, 1:80.

An infectious process was suspected and broad-spectrum antibiotics were given with concomitant use of diuretics, anti-anginal drugs and low dose steroids. On the fourth day after admission the patient developed dyspnea and oxygen desaturation (pO_2 50 mmHg, HbO_2 saturation 80%). Chest X-rays showed increasing bilateral infiltrates. Within a few days the radiological and clinical pictures were compatible with acute respiratory distress syndrome. A pleural tap showed an exudate with a negative culture. Bronchoscopy with bronchoalveolar lavage yielded negative results, and no *Pneumocystis carinii* organisms were seen. He was admitted to the intensive care unit and treated with antibiotics, continuous positive airway pressure and high dose diuretics without further deterioration. On readmission to the internal medicine ward his condition again deteriorated with oxygen desaturation and hypotension (80/60 mmHg), and chest X-rays showed bilateral massive pulmonary infiltrates more on the right lung [Figure 1a]. Administration of vasoactive amines and fluid challenge resulted in a further clinical decline. Intravenous steroids (300 mg hydrocortisone) were given, which had a dramatic effect. After 4 hours the patient felt much better, dyspnea began to resolve and blood pressure rose. On the following day chest X-rays showed resolution of the infiltrates [Figure 1b].

The patient was discharged 3 days later. During the following months the steroids were tapered off without any sequelae.

Comment

Our patient demonstrated the clinical features of post-pericardiotomy syndrome. On admission he had high fever, a pericardial friction rub, pulmonary infiltrates and a high ESR. The lung infiltrates progressed, and only after failure of antibiotics, massive diuretic dose, and preload and afterload reduc-

PPS = post-pericardiotomy syndrome
ARDS = acute respiratory distress syndrome
CABG = coronary artery bypass grafting

ESR = erythrocyte sedimentation rate

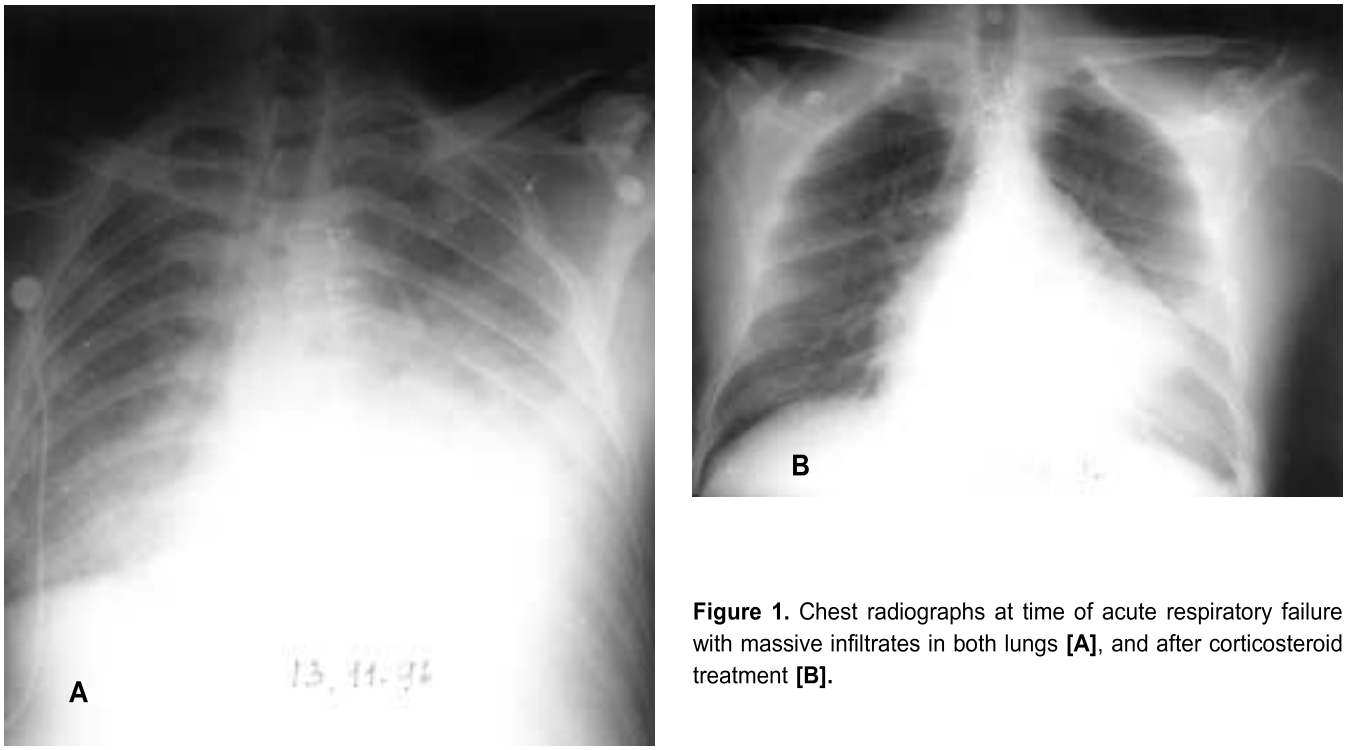

Figure 1. Chest radiographs at time of acute respiratory failure with massive infiltrates in both lungs **[A]**, and after corticosteroid treatment **[B].**

tion therapy, did we consider the possibility of increasing the corticosteroid dosage – with the resulting dramatic response. The diagnosis of acute respiratory distress was made because of the late appearance of massive pulmonary infiltrates (resembling non-cardiogenic pulmonary edema) in PPS.

Non-cardiogenic pulmonary edema after CABG is a rare syndrome, mainly occurring during the procedure or hours to days thereafter [1]. It is thought to be due to an anaphylactic reaction to blood products or to the cardiopulmonary bypass itself, and is responsive to steroid treatment. Kassanoff and Martirossian [2] reported three cases of acute non-cardiogenic pulmonary edema as an early manifestation of post-cardiac injury syndrome, which includes post-pericardiotomy and post-myocardial infarction syndrome. The pulmonary edema occurred hours to 3 days after the cardiac injury, was refractory to diuretics and to preload and afterload reduction, but responded dramatically to corticosteroids.

The incidence of pulmonary infiltrates in post-cardiac injury syndrome varies between 10 and 74% of cases [3,4]. It usually presents within 3 days after presentation and resolves within 48 hours after initiation of corticosteroid therapy [3]. The treatment suggested is administration of anti-inflammatory drugs initially, and only if infiltrates persist should other etiologies be sought [5].

We could not find another case of post-pericardiotomy syndrome manifested as massive pulmonary infiltrates and causing acute respiratory failure 6 weeks after CABG. A possible explanation for the late occurrence of the syndrome is that our patient was on steroid treatment after the CABG, and only when it was tapered to a low dose did symptoms appear. Physicians should be aware that pulmonary insufficiency may be a manifestation of post-pericardiotomy syndrome occurring as late as a few weeks after cardiac injury, since this type of respiratory failure is amenable to corticosteroid treatment.

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Correspondence: Dr. H. Mayan, Dept. of Medicine E, Sheba Medical Center, Tel-Hashomer 52621, Israel. Phone: (972-3) 530-2437, Fax: (972-3) 530-2460
email: mayan@post.tau.ac.il

Winning isn't everything. It's the only thing

Vince Lombardi, American football coach (1913-70)