Postpericardiotomy Syndrome Following Surgery for Nonrheumatic Heart Disease

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Widespread intra cardiac surgery of rheumatic heart disease has brought to medical attention a puzzling postoperative complication, usually designated the "postcommis surotomy syndrome" and often considered to represent reactivation of rheumatic fever. Herein are reported instances of an identical complication following intrapericardial surgery of congenital heart disease. It is suggested that the delayed febrile pleuropericardial reaction represents a traumatic pericarditis and that a preferable name for it is the postpericardiotomy syndrome.

The recent development and widespread application of intra cardiac surgery for patients with rheumatic mitral stenosis has brought to medical attention a frequent postoperative complication, usually designated the "postcommis surotomy syndrome." The nature of this complication is obscure. Its occurrence following mitral valvotomy is believed by some observers to represent a reactivation of rheumatic fever,1-5 while others relate it to the trauma of surgery.6-13

The "postcommis surotomy syndrome" has been reported to occur in 10 to 40 per cent of patients after mitral valve surgery. It is of variable duration and is characterized by pleuropericardial pain, sometimes with effusions, and by slight to moderate fever. The temperature elevation may be continuous with that of the immediate postoperative period or may appear several weeks or months after the operation. The chest pain is usually a constricting precordial pain that frequently radiates to the back, epigastrium, shoulders, or neck and is often accentuated by changes in position, by breathing, or by swallowing. Occasionally there is myalgia or arthralgia but arthritis is rare. Leukocytosis, increased sedimentation rate and elevated C-reactive protein are usually present. The antisera-
had fever during the immediate postoperative period. In 3 patients there was a prolonged febrile course for 12 to 19 days. In 11 patients, fever recurred 1 week to 1 month postoperatively and lasted 3 to 10 days. Two patients were readmitted to the hospital because of the late development of this complication. The temperature during the late febrile reaction varied between 38.5 and 40°C.

Subjective complaints during the time of this postoperative complication were difficult to evaluate in the young children. All the adults and 2 children complained of chest pain during their febrile episode. Frequently the pain radiated posteriorly to the subscapular region and was often aggravated by breathing. Two patients described a feeling of substernal constriction and 2 patients complained of difficulty in swallowing. Ten had an associated nonproductive cough. Two adults complained of generalized muscle aches and pains, but there were no complaints of arthralgia nor were there objective signs of arthritis.

A friction rub was noted in 6 patients during the febrile episode. Chest x-rays at this time demonstrated a temporary increase in cardiac size in all patients. In 7 there was a unilateral pleural effusion, and in 2 it was bilateral. Fluoroscopy in 4 patients revealed diminished cardiac pulsations and a cardiac contour suggestive of pericardial effusion. One such child with marked cardiac enlargement and electrocardiographic evidence of pericarditis developed cardiac tamponade. Pericardial tap disclosed hemopericardium (fig. 3).

The white blood cell count was usually in the range of 10,000 per ml. but it varied between 7,600 and 27,000. The erythrocyte sedimentation rate was elevated in those patients in whom the test was performed. Blood cultures were repeatedly negative. Six patients showed electrocardiographic changes in the S-T segments and T waves characteristic of pericarditis. One adult had 3 episodes of paroxysmal nodal tachycardia during the febrile illness.

The pleural and pericardial effusions resolved spontaneously. Diagnostic taps were performed in only 2 patients. Thoracentesis in 1 man revealed clear, straw-colored fluid that was sterile on culture. Pericardial tap on another patient demonstrated a serosanguineous effusion containing flecks of clotted blood.

Antibiotic therapy did not seem to influence the development or subsidence of the syndrome. All patients received penicillin and streptomycin during the immediate postoperative period. In 10, this therapy was continued or reinstated during the delayed febrile reaction. A different antibiotic was
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added at the time of the unexplained fever in 4 patients. Two patients received no antibiotic therapy. Hormone therapy was not used.

The following cases illustrate the syndrome.

CASE MATERIAL

Case 1. E. R. (NYH 23 75 81) was a 27-year-old white married woman with congenital valvular pulmonic stenosis. Preoperative cardiac catheterization revealed right ventricular pressure of 158/8 mm. Hg. An electrocardiogram showed marked right ventricular hypertrophy and "strain." On June 15, 1956, under hypothermia of 30 C., the pericardium was opened and pulmonary valvotomy was performed through the pulmonary artery under direct vision.

The patient was febrile during the first 4 postoperative days. For the next 2 weeks her temperature was normal but then she developed a fever of 38.2 C. and complained of generalized muscle aches and pains. The white cell count was 8,000 per cu. mm. with 65 per cent polymorphonuclear cells and 13 per cent eosinophils. Sedimentation rate was 52 mm. per hour. Blood culture was negative. An electrocardiogram revealed a sinus tachycardia of 125 per minute and new changes in the T waves and S-T segments. Her fever subsided and she was discharged to her home 3 weeks after operation.

Two days later, she was readmitted because of the development of substernal constriction with radiation to the back. The pain became increasingly more severe and was only temporarily relieved by meperidine hydrochloride. On admission her temperature was 38.5 C., pulse 80 per minute, respirations 26 per minute, and blood pressure 104/66 mm. Hg. Dullness, diminished breath sounds, and a few moist rales were present over the base of the left lung posteriorly. No friction rub was noted. Heart tones were unaltered and a pulmonary systolic murmur was present. Chest x-ray revealed an enlarged heart and pleural effusion on the left side (fig. 1). An electrocardiogram was essentially unchanged from that recorded 1 week earlier. The hemoglobin was 11.5 Gm. and the white cell count 8,400 per mm.³. Four blood cultures were negative.

The patient was febrile for 4 days. Her temperature occasionally rose as high as 39.5 C. No antibiotic therapy was given and the symptoms and signs subsided spontaneously. She was discharged with a diagnosis of "postvalvotomy syndrome." There has been no recurrence.

Case 2. P. T. (NYH 70 80 01) was an 18-year-old white male who was operated on for congenital valvular pulmonic stenosis. Preoperative cardiac

Fig. 1. Top. Preoperative film in case 1. Middle. Chest x-ray 1 month following surgery shows increase in cardiothoracic ratio and left pleural effusion. Bottom. Five weeks postoperatively there is decrease in heart size and residual pleural effusion.
catheterization revealed a right ventricular pressure of 153/13 mm Hg. On June 17, 1955, a pulmonary valvotomy was performed via a transventricular route at normal body temperature.

The patient was febrile during the first week postoperatively. On the tenth postoperative day he complained of a severe, crushing, substernal pain, which was accentuated by deep breathing. Electrocardiogram revealed S-T elevation in leads I and II, consistent with pericarditis. Chest x-ray revealed an increase in heart size and hazy density over the lower left lung. He remained afebrile during this period and the pain subsided spontaneously after 3 days. He was discharged on the twenty-fourth postoperative day with a diagnosis of postoperative pericarditis.

Three days following discharge, he was readmitted with a fever of 39.5 C and chest pain of 30 hours' duration. The pain was substernal and was characterized as a dull, aching feeling of constriction that radiated to the back and right shoulder. A pericardial friction rub was present and there were dullness and rales at the base of the left lung. Chest x-ray again showed an enlarged heart and left pleural effusion (fig. 2). Changes consistent with pericarditis were still present in the electrocardiogram. The white blood cell count was 10,800, with 84 per cent polymorphonuclear cells. Three blood cultures were sterile. Thoracentesis yielded 200 ml. of clear, straw-colored fluid.

He was given penicillin and streptomycin and when he failed to improve, erythromycin was added. He was febrile for 10 days. Antibiotics were then discontinued and the patient was discharged. There has been no recurrence of symptoms.

Case 3. T. T. (NYH 73 86 66) was a 6½-year-old white boy who was explored for a possible patent ductus arteriosus. Preoperative cardiac catheterization revealed a significant left-to-right shunt into the pulmonary artery. Some increase in pulmonary vascular resistance and moderate elevation of pulmonary artery pressure were noted. On July 13, 1956, an exploratory thoracotomy was performed. A ductus was not present. Therefore the pericardium was incised widely and the base of the heart explored. The findings were characteristic of an aortic-septal defect. No further surgical procedures were attempted.

The child had a stormy postoperative course. A high, spiking fever up to 40 C persisted for 19 days, despite intensive antibiotic therapy. Repeated blood cultures were negative. The white cell count rose as high as 27,000. He developed signs of pericardial effusion and of cardiac tamponade with increasing heart size, friction rub, marked damping of heart sounds, and an enlarging, tender liver. Temporary improvement followed digitalization. Dullness, diminished breath

![Fig. 2. Top. Preoperative chest x-ray in case 2. Middle. One month after surgery there is marked increase in transverse cardiac diameter and pleural effusion on the left. Bottom. Resolution of pleuro-pericardial effusion 3 months postoperatively.](image-url)
sounds, and rales were present at the left base. Chest x-ray showed marked cardiac enlargement and left pleural effusion (fig. 3). Serial electrocardiograms revealed changes in the S-T segments and T waves, and decreased amplitude of the QRS complexes consistent with pericarditis with effusion. Diagnostic pericardioentesis on the thirteenth postoperative day yielded serosanguineous fluid containing clotted blood.

The patient gradually improved and was no longer febrile after the third week. He has returned to his preoperative level of activity and has experienced no recurrence of symptoms.

**DISCUSSION**

Thirteen patients with congenital heart disease have developed this syndrome following cardiac surgery which involved opening the pericardium. The cardiac lesions included valvular and subvalvular pulmonic stenosis, atrial septal defect, total anomalous pulmonary venous drainage, congenital mitral stenosis, and aortic septal defect. One of the authors has observed such a reaction in patients with tetralogy of Fallot when the pericardium was entered to locate a small pulmonary artery.

Milstein and Brock have described a similar syndrome during the second and third weeks following pulmonary valvotomy for congenital pulmonic stenosis. They did not comment on its similarity to the “postcommissurotomysyndrome” but remarked that it appeared to be due to a noninfective pericarditis. Bedford and associates and Likoff (whose personal communication was reported by Elster et al. and Papp and Zion) noted a similar complication following closure of atrial septal defects. Cooley has encountered this reaction in patients with ventricular septal defects whose pericardium was opened to permit closure of the defect. Larson reported a similar complication following chest surgery on nonrheumatic patients. However, a review of available literature failed to disclose documented reports of the syndrome following surgery for congenital heart disease.

We prefer to designate the delayed postoperative occurrence of fever and chest pain, often associated with evidence of pericardial or pleural involvement, as the postpericardiotomy syndrome.

**Fig. 3.** Top. Preoperative film in case 3. Note evidence of left atrial and left ventricular enlargement and pulmonary vascular engorgement. Middle. Striking increase in cardiac silhouette is present 9 days after operation. Left base is obscured. Bottom. Note improvement since previous film, 3 1/2 weeks following surgery. Later return to preoperative picture.
diotomy syndrome rather than the frequently used term “postcommisurotomy syndrome.”

Valvotomy, cardiotomy, and incident injury to cardiac muscle as responsible factors in the syndrome seem to be excluded, since the complication appeared in 4 patients with exploratory pericardiotomy in whom no further surgical procedures were attempted, and it followed valvotomy on the pulmonary valve by both the transventricular and the pulmonary artery approaches.

In the patients with both rheumatic and nonrheumatic heart disease in whom this complication has developed, pericardial incision was performed. The postoperative syndrome then occurred with like frequency, whether or not the additional surgical procedures involved entering the left atrium for mitral valvoplasty, the right atrium for atrial septal defect closure, the right ventricle for transventricular pulmonary valvotomy or closure of ventricular septal defects, the pulmonary artery for pulmonary valvotomy, or intrapericardial exploration alone for an inoperable lesion.

It is of interest that 5 patients with surgery for pericardial cysts did not develop this complication. In each instance, the cyst communicated with the pericardial cavity through a narrow base which was clamped and cut at the time of operation. The pericardial cavity was not entered and the possibility of intrapericardial bleeding was minimal.

Although other factors may be important in the development of this postoperative complication, it would appear that the syndrome represents a traumatic pericarditis, possibly associated with oozing of blood into the pericardial cavity. Trauma to the pericardium as an etiologic factor has also been suggested by Elster and associates.8 Dressler3 noted the striking similarity between acute benign pericarditis and the postpericardiotomy syndrome. The clinical triad of pericarditis, pleuritis, and pneumonitis are characteristic of both groups, as are the frequent relapses and the subsequent benign course. Nevertheless, it was his impression that the “postcommisurotomy syndrome” represented a reactiva-

tion of rheumatic fever, since it was a complication that had not yet been reported following surgery in congenital heart disease.

Soloff and Zatuchnic19 observed moderate to massive cardiac enlargement following mitral commissurotomy in 44 patients. They believed this represented a reactivation of rheumatic fever in most instances. However, the possibility that their patients also suffered from a traumatic pericarditis is suggested by the fact that in the 2 patients in whom pericardiocentesis was performed, serosanguineous fluid was found. Janton and coworkers10 reported 7 patients who required pericardial taps following mitral valve surgery. In each instance serosanguineous fluid, which often contained clotted blood, was obtained.

All our patients recovered without demonstrable sequelae during 1 to 9 years of postoperative observation. However, if extensive bleeding has occurred into the pericardial cavity, it is possible that some patients may develop constrictive pericarditis. Lazlo20 described a patient on anticoagulant therapy who developed a hemopericardium and cardiac tamponade. Eight months later, an operation was necessitated because of constrictive pericarditis. Similar complications have been reported as a late development following nonpenetrating injuries to the chest and have been attributed to a traumatic hemopericardium associated with the chest injury.20-28 For example, Ada and associates21 performed a pericardial resection in a patient who was found to have old blood in the pericardium and who had sustained 2 nonpenetrating chest injuries, 2 years and again 3 months before the operation.

It has been suggested that fibrinolytic enzymes be used in patients with hemopericardium to prevent subsequent development of constrictive pericarditis.28 Such therapy may be of value in patients whose pericardium is opened and traumatized during surgery.

**Summary**

A syndrome indistinguishable from the “postcommisurotomy syndrome” has been
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observed among patients with congenital malformations of the heart who survived intrapericardial surgery. It appeared as frequently (13 of 24 pericardiotomies) as has been reported in rheumatic patients who recovered from mitral valve surgery (10 to 40 per cent). The syndrome developed following transventricular and transarterial valvoplasty for pulmonary stenosis, closure of septal defects, and exploration of the pericardium for inoperable congenital cardiac lesions. Although other factors may be important in the pathogenesis of this condition, the feature common to these operations in the nonrheumatic as well as rheumatic patients was wide incision of the pericardium. This postoperative complication was not noted after other operations for congenital heart disease where-in the pericardium was not disturbed, nor was it found in patients where a small segment of pericardium was clamped to permit the removal of a pericardial cyst.

Since the condition appeared after pericardial incision, with or without cardiotomy or valvotomy, the term “postpericardiotomy syndrome” is suggested as more universally applicable for this postoperative complication.

The syndrome is interpreted as a traumatic pericarditis, possibly a reaction to blood in the pericardial sac. The occurrence of this postoperative manifestation in nonrheumatic subjects is a compelling argument against the concept that the syndrome in patients with mitral valvotomy usually represents reactivation of rheumatic fever.

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SUMMARIO IN INTERLINGUA

Un syndrome, quo es indistinguibile ab le syndrome postcommissurotomico, esseva obserbata in patientes con congenite malformaciones del corde qui supervivivaa a operationes intrapericardial. Iilo appareva tanto frequentemente (in 13 ex 24 pericardiotomias) como, secundo le reportos, in patientes rheumatici qui se restabliva ab operationes del valvula mitral (10 a 40 pro cento). Le syndrome se disveloppava post valvoplastia transventricular e transarterial pro stenosis pulmonar, post clauzion de defectos septal, e post exploration del pericardio in casos de inoperabile congenite lesiones cardiac. Ben que altere factores es possibilemente importante in le pathogeneese de iste condition, le aspecto commun de iste operationes in le patientes tanto nonrheumatic como etiam rheumatic esseva un large incision del pericardio. Iste complication postoperatori non esseva note post operationes pro congenite morbo cardiac in le quales le pericardio non esseva disturbate; similemente illo non esseva trovate in patientes in qui un micre segmento del pericardio esseva crampate pro permetter le excision de un cyste pericardial.

Proque le condition appareva post incision pericardial, con e sin cardiotomy o valvotomy, le termino “syndrome postpericardiomyotic” es proponite como plus universalmente applicabile a iste complication postoperatori.

Le syndrome es interpretate como un pericarditis traumatic, possibilemente un reaction al presentia de sanguine in le sacco pericardial. Le occurrencia de iste manifestation postoperatori in subjectos non-rheumatic es un argumento convincente contra le notion que le syndrome in patientes con valvotomy mitral representa usualmente un reactivation de febre rheumatic.

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