SECTION ON
CARDIOVASCULAR DISEASES

Occluding Thrombi of the Left Atrium

Report of Four Cases Treated Surgically*

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Fragmentation of an atrial thrombus with embolization is a serious and frequently disastrous complication of mitral valvuloplasty. Fortunately, the majority of thrombi are well-organized and firmly attached to the atrial wall or to the appendage. If care is taken not to dislodge any adherent clot, satisfactory valvuloplasty ordinarily can be performed by a closed method with a respectably low incidence of operative embolism. In contrast, a free-floating or pedunculated intracavitary thrombus, although less common, poses a formidable problem in surgical management and judgment.

"Ball thrombus" was a name coined by Wood¹ (1814) to describe an organized, unattached clot whose cross sectional diameter was greater than that of the orifice of the chamber containing it. The term has subsequently been used to include pedunculated thrombi also. These thrombi are usually found in the fibrillating left atria of patients with tight mitral stenosis. Their size varies from one cm. in diameter to large masses filling the greater part of the left atrial cavity. Although these thrombi are usually well-organized, they are often partially covered with recent friable clot. Occluding thrombi, whether free-floating or pedunculated, aggravate symptoms of mitral stenosis or cause death by one or any combination of three mechanisms: (1) intermittent occlusion of an already compromised mitral orifice; (2) impaired left atrial filling; or (3) arterial embolism. The lesion would appear to be relatively uncommon. Wallach and his associates⁶ found 16 occluding left atrial thrombi among 509 patients with mitral stenosis studied at necropsy, an incidence of 3.1 per cent.

This report is concerned with four cases of occluding left atrial thrombi encountered among 105 operations performed for the relief of mitral stenosis at the Rhode Island Hospital.

Case Reports

Case 1: V. M., 52 year-old man, deaf-mute, was admitted to the Rhode Island Hospital on April 27, 1957 with mild congestive heart failure. There was no history of rheumatic fever or previous arterial emboli. His illness began six years earlier with increasing fatigability and exertional dyspnea. He was treated with digitalis, and subsequently was admitted to another hospital on two occasions because of syncope and chest pain. On each of these admissions, cyanosis of his face, hands and feet was noted.

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Recovery occurred promptly each time. He had had atrial fibrillation for two years. Three months before this admission, a severe episode of congestive heart failure required hospitalization.

On examination, blood pressure was 110/80 mm Hg. The pulse was 74 and totally irregular. The neck veins were moderately distented. There was edema of the lower extremities. A low-pitched Grade II rumbling diastolic murmur was audible over the apex. The pulmonic second sound was accentuated. The first sound at the apex was distinct. Rales were heard over both lung bases. All peripheral pulses were easily felt. Fluoroscopy showed an enlarged right ventricle; there was no evidence of calcification in the mitral valve region. An electrocardiogram revealed auricular fibrillation with digitalis effect.

The patient responded satisfactorily to a strict medical regimen and he was considered a good candidate for mitral valvuloplasty. An occluding thrombus was not suspected. Eleven days after admission, a thoracotomy was performed through the left fourth intercostal space. The major branches of the aortic arch were encircled with loops of umbilical tape which were used to occlude blood flow during all intracardiac maneuvers. A circumferential purse string of heavy silk was placed around the base of the left atrial appendage. The tip of the appendage was amputated and the atrium was flushed in order to wash out any free thrombi. None was recovered. Whole blood was rapidly infused during this period to compensate for the blood loss. When a finger was introduced into the left atrium, a firm spherical mass, measuring approximately 2.5 cm in diameter, was felt (Fig. 1). It was attached by a thin pedicle to the posterior atrial wall immediately above the mitral valve orifice. With each ventricular diastole, the mass was felt to enter the depression over a tightly stenosed mitral orifice. During exploration, the thrombus became detached. Several unsuccessful attempts were made to trap the thrombus against the atrial wall with the finger. The mitral valve deliberately had not been opened and the intracardiac finger was withdrawn. During the intra-atrial manipulations, a small tear had occurred in the lateral wall of the atrial appendage proximal to the purse string suture. The appendage was unusual in that it was short and was connected to the atrium by a broad neck. The auricular clamp was then released and the escaping blood, under pressure, effectively delivered the thrombus into the appendage, where it became impacted. Gentle dilatation of the already enlarged opening in the atrial appendage resulted in the thrombus being expelled. The atrium was "flushed-out" again and a satisfactory valvuloplasty was subsequently performed. Examination of the thrombus revealed it to be roughly spherical in shape and firm and rubbery in consistency. Microscopically, it was relatively well-organized except for a peripheral zone of recent clot. The patient made an uncomplicated recovery and was discharged from the hospital on his 16th post-operative day. He has been followed for four years during which time he has had no further episodes of chest pain, syncope or pulmonary edema.

Case 2: A. M., 53 year-old woman, admitted to the Medical Service of the Rhode Island Hospital on April 20, 1958. She complained of pain and numbness of both legs of six hours duration. These symptoms had occurred abruptly, while she was preparing

FIGURE 1: Artist's drawing of the occluding thrombus encountered in Case 1.
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FIGURE 2:
Technique employed to enlarge the atrial opening and "flush-out" the free-floating intracavitary thrombus.

FIGURE 2A

FIGURE 2B

FIGURE 2C
dinner, and had been associated with transient lower abdominal pain and vomiting. She had had rheumatic fever at age 15. Atrial fibrillation together with congestive heart failure had necessitated hospitalization six years before this admission. Digitalis and the intermittent use of diuretics controlled cardiac decompensation, but atrial fibrillation persisted. Four years later she sustained an embolic occlusion of the left brachial artery and a questionable splenic infarction. She did not receive anticoagulant therapy. Subsequently she noted increasing dyspnea and orthopnea.

On examination, the blood pressure was 120/86. The heart rate was 82 and the rhythm grossly irregular. The left lower extremity was cold, mottled and cyanotic. The left femoral pulse was absent. The right leg was cool and slightly mottled. The right femoral pulse was faintly palpable, but the right popliteal pulse was absent. Auscultation of the heart revealed a Grade II apical diastolic murmur and a Grade I systolic murmur along the left sternal border. The pulmonic second sound was accentuated. Except for a moderate elevation of the white-cell count, routine laboratory data were not remarkable. An electrocardiogram confirmed the existence of atrial fibrillation. Fluoroscopy showed an enlarged right ventricle and calcification in the mitral valve region.

Soon after admission, all evidence of ischemia of the right leg disappeared. The appearance of the left lower extremity remained unchanged. Despite a surgical recommendation for prompt embolectomy, the Medical Service elected to anticoagulate the patient and manage the embolism "conservatively." The arterial circulation in the limb improved in the next 24 hours and three days later it was clear that the viability of the left leg had been preserved. After three weeks of careful management with digitalis, mercurials, salt restriction and anticoagulants, the medical attitude became more aggressive and the patient was referred for mitral valvuloplasty. The surgical procedure was undertaken in precisely the same manner as in the case described before, including isolation of the aortic arch branches with umbilical tape. Preliminary flushing of the atrium did not yield any thrombi. Finger exploration of the atrium revealed an unattached irregularly lobulated oval mass which, when later studied, measured 4 x 3 cm. It seemed reasonable to attempt to evacuate the thrombus through an enlarged incision in the atrial wall. A row of opposing mattress sutures was placed in the lateral wall of the atrial appendage and the atrial wall. When traction was applied to these sutures, the appendage and a segment of the lateral atrial wall were elevated and grasped with an atraumatic occluding clamp (Fig. 2A). A 2 cm. incision was made between the opposing sutures beginning at the amputated tip of the appendage. Whole blood was rapidly administered through two venous cannulae. The occluding clamp was released briefly and the thrombus was expelled with the ensuing gush of blood (Fig. 2B). During this maneuver, an assistant maintained continuous traction on stay sutures to ensure that the atrial wall beyond the incision would again be engaged when the occluding clamp was closed. The blood loss, while evacuating the thrombus, was estimated to be between 300 and 400 cc. The incision in the atrium was then carefully closed with a running suture of silk reinforced with interrupted sutures of the same material (Fig. 2C). In spite of calcification of the leaflets, a satisfactory valvuloplasty was accomplished and the patient's recovery was uneventful. In the two years since operation, her cardiac symptoms have improved. However, she has continued to have intermittent claudication in the left lower extremity.

FIGURE 3: Photograph of the atrial thrombus in Case 2.
Examination of the thrombus showed it to be firm and rubbery, composed of two bulbous extremities connected by a narrow waist (Fig. 3). Histologic examination showed it to be organized in many places and mixed with clot of varying age.

Case 3: I. A., 54 year-old woman, was admitted to the Rhode Island Hospital on February 5, 1959 for definitive surgical treatment of mitral stenosis. She had had a heart murmur for 24 years, but could not recall having had rheumatic fever. Three years previously she had developed atrial fibrillation and had been treated with digoxins, diuretics and salt restriction, but nevertheless had frequent attacks of paroxysmal nocturnal dyspnea. Three months before this admission, she had been seen, as an emergency, by the Medical Service because of severe substernal pain and dyspnea. These symptoms disappeared abruptly without specific treatment. She had suffered no previous episodes of embolism.

Examination disclosed a thin, middle-aged woman whose positive physical findings were referable to her cardiovascular system. Blood pressure was 140/90 mm.Hg. in both arms. A Grade II diastolic, crescendo murmur was heard at the apex. A softer and inconstant systolic murmur was audible to the left of the sternum in the fourth interspace. The pulmonic second sound was accentuated and split. All peripheral pulses were present. The vital capacity of the lung was 2.1 liters. An electrocardiogram showed auricular fibrillation with a ventricular rate of 70. Fluoroscopy revealed a moderately enlarged heart with calcification in the mitral valve region.

On February 23, 1959, an operation similar to the one previously described was performed. A firm, unattached thrombus, measuring 3.5 x 3.5 cm. was found. It was evacuated through a controlled atriotomy with the loss of approximately 400 cc. of blood. This was poorly tolerated and an alarming bradycardia developed. Blood pressure fell to 70/30 mm.Hg. After a period of five minutes, during which whole blood was rapidly infused, cardiac function improved. The mitral valve was tightly stenosed and valvuloplasty was performed with difficulty. Satisfactory incision of both commissures was accomplished, but extensive calcification precluded good mobilization of the valve cusps. Retained bronchial secretions with atelectasis complicated an otherwise satisfactory postoperative course. The patient was discharged from the hospital two weeks following operation. Seventeen months have elapsed since operation and the patient has had no episodes of paroxysmal nocturnal dyspnea. No late embolism has occurred.

Examination of thrombus showed it to be roughly conical in shape. An indented ridge at the tip conformal to the size of the stenosed valve orifice (Fig. 4). The histologic picture was varied. The base of the thrombus was well-organized and covered with endothelium. Peripherally, there was a mixture of clot of varying age showing stratification.

Case 4: L. M., 53 year-old woman, admitted December 6, 1959 with intractable cardiac failure. She was known to have had mitral stenosis for 43 years. She had had

FIGURE 4: The thrombus recovered from Case 3.
three uncomplicated pregnancies and had been relatively free of symptoms until one and one-half years before admission. At that time, dyspnea, cough and peripheral edema responded to treatment with digitalis, mercurials and salt restriction. Three months before admission, an effort to anesthetize her for the removal of several carious teeth was frustrated by the development of severe cardiac arrhythmias. Later she developed atrial fibrillation, a dry, nonproductive cough, and recurrent edema refractory to full doses of digitalis and diuretics. In spite of increasing dyspnea, it was of interest that the patient obtained maximum relief in her breathing by lying prone in bed. All other positions, except sitting upright, resulted in severe dyspnea and paroxysms of coughing.

Examination disclosed a small, thin, chronically ill woman lying on her abdomen in bed. Her nose, fingers and toes were cyanotic. Her neck veins were distended. There were rales at both lung bases. The heart was enlarged to the left. Blood pressure was 115/80 and the heart rate was 100 and totally irregular. A loud, high-pitched, squeaking murmur was heard along the left sternal border. A softer diastolic murmur was heard over the apex. The second sound in the pulmonary area was accentuated. The liver was felt five finger-breathths below the costal margin. There was pitting edema of the sacrum and the lower extremities. An electrocardiogram showed fibrillation with right ventricular hypertrophy. Fluoroscopy disclosed an enlarged heart with a prominent pulmonary conus, evidence of calcification in the region of the mitral valve, and bilateral pleural effusions.

Her symptoms became worse in spite of intensive medical treatment, and it was the consensus that her prognosis was hopeless without surgical treatment. Open operation was elected, since her mitral valve was known to be extensively calcified and the clinical findings suggested the presence of an occluding intra-atrial thrombus. In preparation for the definitive cardiac operation, tracheotomy was performed with the patient in the sitting position. On December 19, 1959, right thoracotomy through the bed of the resected fifth rib was performed. The superior vena cava was cannulated through the right atrial appendage and the inferior vena cava from below, through an incision in the right common femoral vein. Arterial return was through a stainless steel cannula introduced into the right femoral artery. Total heart-lung by-pass was carried out for one hour and 56 minutes, using a heart-lung machine which we have previously described.3 The left atrium was opened widely posterior to the interatrial sulcus. A pedunculated thrombus, measuring 3 x 3 x 4 cm. was found attached to a thrombus filling the left atrial appendage (Fig. 5). The mitral valve was extensively calcified and tightly stenosed. A gauze sponge was packed into the stenosed valve orifice to trap any thrombotic fragments and the friable thrombus was evacuated in several pieces. The orifice of the atrial appendage was then closed from within the atrium with a running suture of silk. A mitral commissurotomy was achieved under direct vision with the use of appropriate instruments. Although both the anterior and posterior commissures were adequately incised to the valve annulus, dense calcification in the region of the posterior commissure rendered the valve leadets immobile. An

FIGURE 5: Drawing showing the thrombus as it appeared to the surgeon at open-operation.
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edematous atrial wall was closed with difficulty. After the circulation had been re-established, it was necessary to return to partial cardiopulmonary by-pass several times in order to achieve a watertight seal. She tolerated the procedure well and was awake, responding to questions, as the last skin sutures were placed. Her postoperative course was gratifying in the first 24 hours. She was alert and cooperative. The serum electrolytes and pH showed no deviation from normal. Venous pressure varied from 10 to 15 mm. Hg, and systolic arterial blood pressure remained at 100 mm. Hg. Early on her second postoperative day, however, she developed digital cyanosis with a progressive fall in blood pressure despite an adequate blood volume and normal blood chemistry. Death occurred abruptly 48 hours following operation. Necropsy revealed no obvious anatomic cause of death. All suture lines were intact. The heart was enlarged, pale and flabby. Microscopically, the left ventricular myocardium was infiltrated with fat. The cause of death was considered to be due to chronic left ventricular insufficiency.

Discussion

An accurate clinical diagnosis of an occluding intra-atrial thrombus complicating tight mitral stenosis is rarely made ante-mortem. The clinical picture is usually one of severe mitral stenosis. Several clinical clues including peripheral cyanosis, episodes of syncope, frequent attacks of paroxysmal dyspnea, ischemic chest pain, changing murmurs and relief of dyspnea in the knee-chest or prone positions have been described. In retrospect the signs and symptoms in the first three patients described above suggested, but were not diagnostic of intermittent ball-valve occlusion of the mitral valve. The clinical course, peripheral cyanosis and the postural relief of dyspnea encountered in Case 4 resulted in an accurate clinical diagnosis being made. Angiocardiography as described by Steinberg will demonstrate intra-atrial thrombi and is perhaps the most suitable method of making a correct pre-operative diagnosis. Routine use of angiocardiography, however, in the pre-operative evaluation of the severely ill patient with tight mitral stenosis, chronic atrial fibrillation and/or a history of previous embolism, is debatable.

The methods employed in removing intracavitary thrombi, encountered unexpectedly during mitral valvuloplasty, depend, to a great extent, upon the size and age of the thrombus. Clearly, a large ball thrombus requires an open technique, using hypothermia or a pump-oxygenator for its safe removal. On the other hand, small (2 cm. in diameter) clots usually be “flushed-out” through the atrial opening in the atrial appendage. Firm thrombi of intermediate size, however, constitute a problem. Class IV patients with tight mitral stenosis tolerate operation poorly unless a reasonably satisfactory opening in the mitral valve is achieved. Thus, when an occluding thrombus is found unexpectedly during the closed operation for mitral stenosis, it would appear reasonable to attempt to “flush out” the thrombus and proceed with an effective mitral commissurotomity. It is of interest that among 29 ball thrombi collected by Radding, 20 ranged from 2.5 to 3.5 cm. in diameter. This would indicate that a significant number of intracavitary thrombi might be removed by methods less dangerous than caval occlusion. For extracting soft intracavitary clot, Julian advocated the use of strong suction through a glass tube. He did not recommend this method, however, where an organized thrombus might occlude the aspirator and become fragmented. Jamison and his associates described a method whereby the thrombus was grasped with a Kelly clamp and withdrawn through the atrial appendage. They did not mention how often fragmentation occurred. Some authors have described performing a mitral valvuloplasty before dealing with the intracavitary thrombus. This practice would appear to be unwise because the thrombus might escape into the ventricle where it could fatally occlude the outflow tract, or embolize to the aortic bifurcation. The technique employed in the first three patients described above was chosen to minimize the hazard of fragmenting the thrombus. Its effectiveness depended upon the presence of a tight mitral valve which confined the thrombus to the atrium and resulted in an elevated atrial pressure sufficient to eject the thrombus during the brief period the atriotomy was open. Bailey and others have shown that a short incision in the atrial wall may be made with reasonable safety if it is controlled with appropriately placed stay or mattress sutures. A short incision (never longer than 2 cm.) extending from the waist of the appendage into the atrial wall enlarged the atrial opening enough to permit passage of the thrombus in two of the patients described. The size of the thrombus and the configuration of the atrial appendage in the first patient simplified the technical problem and blood loss was minimal. In the third patient, a larger atrial opening resulted in an acute blood loss. This patient tolerated hemorrhage poorly and her condition was precarious for several minutes after the thrombus had been removed. Rapid transfusion of blood through two large venous portals, however, quickly restored the circulating blood volume. Admittedly, sudden alterations in blood volume are not well tolerated by patients with severe mitral stenosis. However, in evaluating the overall risk, the undesirable effects of transient hypovolemia are, at least, subject to prompt treatment. On the other hand, a shower of emboli from a fragmented thrombus is a disaster in which the surgeon finds himself helpless. In the fourth patient, where the presence of an occluding intra-atrial thrombus was anticipated, an open method...
utilizing cardiopulmonary by-pass was elected. The technical facility in dealing with the intracardiac thrombus under direct vision convinced us of the superiority of this method, and we would employ an open technique again when an occluding thrombus was suspected. Merendino has recently pointed out that the mortality for Class IV patients undergoing direct vision commissurotomy is rapidly approaching that of the closed method. It would appear that the day is near when all patients suspected of having any intracardiac clot, e.g., patients having atrial fibrillation, with or without a history of previous embolization, may be more effectively treated with an open operation.

SUMMARY

Among the morphologic variants of left atrial thrombi, the unattached ball or pedunculated thrombus poses the greatest problem in surgical management and judgment. Clearly, large occluding thrombi require an open technique for their safe removal. On the other hand, a number of intermediate-sized thrombi can be treated surgically by less elaborate means when encountered unexpectedly. Four clinical cases encountered among 105 operations at the Rhode Island Hospital are presented to illustrate the problem in diagnosis and surgical management. The surgical technique employed in three patients in whom free-floating intra-atrial thrombi were encountered unexpectedly was chosen in an effort to avoid fragmenting the thrombus. It took advantage of a tight mitral stenosis which effectively restricted the thrombus to the atrium and created an elevated atrial pressure sufficient to expel the thrombus through an enlarged but suture-controlled atriotomy. A fourth patient was suspected of having an occluding thrombus and underwent an open operation.

RESUMEN

Entre las variantes morfológicas de la trombosis auricular izquierda, el trombo esférico o pedunculado presenta el problema mayor en el tratamiento quirúrgico y en la decisión.

Claramente, los trombos grandes oculosis requieren una técnica abierta para su extracción segura. Por otra parte, cierto número de trombos de tamaño intermedio, pueden ser tratados quirúrgicamente por procedimientos menos complicados cuando se encuentran inesperadamente. Cuatro casos clínicos encontrados entre 105 operaciones por estenosis mitral en el Hospital Rhode Island se presentan para ilustrar el problema del diagnóstico y del tratamiento. La técnica quirúrgica empleada en tres enfermos en quienes había trombos libremente chocando dentro de la cavidad auricular se escogió para evitar la fragmentación del trombo. Se aprovechó la ventaja de que había una estenosis marcada que restringía la posición del trombo a la aurícula y creaba una presión suficiente para expulsar el trombo a través de una atriotomía grande pero controlable por la sutura. Un cuarto enfermo fue sospechoso de tener un trombo oclusivo y se trató por operación a cielo abierto.

RESUMÉ

Parmi les variantes morphologiques de thrombus auriculaires gauches, le thrombus libre en forme de balle et le thrombus pédiqué posent le plus grand problème dans la conduite chirurgicale et l’appréciation. Clairement, des thrombus volumineux faisant obstruction demandent une technique à cœur ouvert pour que leur exérèse puisse être réalisée sans danger. D’un autre côté, un nombre de thrombus de taille intermédiaire peuvent être traités chirurgicalement par des procédés plus simples quand on les rencontre d’une façon inattendue. Quatre cas cliniques rencontrés sur 105 opérations de sténose mitrale à l’Hôpital de Rhode Island sont présentés pour illustrer le problème du diagnostic et de la conduite chirurgicale. La technique chirurgicale utilisée pour trois malades chez lesquels on rencontre d’une façon inattendue des thrombus intra-auriculaires flottant librement fut choisie de façon à éviter la fragmentation du thrombus. Elle tira parti d’une étroitesse sténosée mitrale qui limita effectivement le thrombus et créa une élévation de la pression artérielle suffisante pour expulser le thrombus à travers une atriotomie large mais contrôlée par la suture. Un quatrième malade fut soupçonné d’avoir un thrombus oclusif, et fut soumis à une opération à cœur ouvert.

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REFERENCES


PULMONARY EMPHYSEMA: ETIOLOGIC FACTORS AND CLINICAL FORMS

Chronic pulmonary emphysema is considered in terms of the major etiologic forces producing this disease. Three basic forms are described: a) chronic bronchitis and emphysema, b) pulmonary fibrosis and emphysema, and c) nonobstructive bullous emphysema.

Within the category of chronic bronchitis and emphysema, two chief clinical forms can be distinguished: a) obstructive emphysema with ventilatory insufficiency, and b) obstructive emphysema with ventilatory insufficiency and alveolar hypoventilation.

In the emphysema associated with pulmonary fibrosis, the basic etiologic forces are: a) traction, and b) obstruction.

The class of primarily nonobstructive bullous emphysema is presented as a disease in which the etiologic factor is an essential destruction or "atrophy" of respiratory tissues. Secondary air passage obstruction, however, occurs from various mechanical causes, and is an important and almost inevitable complication of this disease.