flows for the expulsive phase of the cough. With the expulsive phase, the glottis opens and the high-pressure gradient generates rapid airflow. Both inspiratory and expiratory muscles are actively involved in coughing, and extreme changes in intrapleural pressure occur due to active contraction of these muscles. This intrathoracic pressure swings as well as repeated cough results in multiple complications.

Numerous complications are associated with coughing. These include syncope, rupture of subconjunctival nasal and anal veins, bradycardia, pneumomediastinum, pneumomediastitis, hematoma, incontinence, the rupture of muscles, herniation of the lung through the intercostal space, and the fracture of ribs. The fracture of ribs can be explained by either of two different theories. The first mechanism of rib fracture in cough is similar to that of stress fractures. When force (muscle contraction) is applied to an object (a rib), the object is subjected to stress. The stress will cause deformation of the object. When the deformation exceeds the elastic limit of the object, it undergoes inelastic deformation. Repeated trauma, as in paroxysms of cough, can produce inelastic deformation in the most vulnerable part of the ribs, the middle third. This will result initially in minor cracks of the ribs and later, as the trauma continues, in fractures. Fractures can occur in any rib, but the ones most commonly involved are the fifth to 10th ribs.

The second mechanism of rib fracture may be due to opposing muscle forces acting on the ribs. The diaphragm is mainly an inspiratory muscle. The costal part of the diaphragm is attached to the lower six ribs and their cartilage. The muscles of expiration are the chest wall muscles, which include the internal intercostals, the triangularis sterni, the serratus posterior, the quadratus lumborum, and the abdominal muscles (including the external and internal oblique, the rectus abdominis, and the diaphragm). The diaphragm also acts as an expiratory muscle during activities requiring high intrathoracic pressure like coughing, vomiting, and sneezing. This expiratory activity of the diaphragm is related directly to the intrapleural pressure and follows the expiratory activity of the transversus abdominis muscle. It is speculated that the diaphragmatic contraction will help to stabilize the thoracic cavity during the expulsive phase of cough. The study by Nakadi and Vanderhoeft described a fracture line starting from a point 4 cm from the costochondral junction of the fourth rib running obliquely caudal and laterally to the ninth rib in the midaxillary line. This line falls on the muscular attachments of the external oblique and serratus anterior muscles. The opposing actions of these muscles on the same ribs can result in fractures. Simultaneous contraction of the shoulder girdle muscles, especially of the serratus anterior, also contributes to the rib fractures by pulling the ribs upward and laterally while the abdominal muscles pull the ribs medially and downward. The most common cause of diaphragmatic rupture is trauma. Seven percent of thoracic injuries and 22% of thoracoabdominal injuries are associated with diaphragmatic injury. Left-sided ruptures are five times more common than right-sided injuries. During forced respiratory movements, the muscles of the abdominal wall contract pushing the diaphragm upward and the ribs inward and downward. A sudden and forceful Valsalva maneuver can result in the lack of coordination of different muscles of expiration. This can contribute to the rupture of the diaphragm. Since both the diaphragm and abdominal muscles are attached to the lower ribs, this kind of opposing action can result in rib fractures. In the presence of existing rib fractures, antagonistic actions of muscles on different fragments of ribs probably contribute to the tearing of the diaphragm.

Our patient had paroxysmal coughing as a result of bronchitis and sustained rib fractures that resulted in flail chest and rupture of the diaphragm, which required surgical intervention. To our knowledge, this is the first report of diaphragmatic rupture as a result of paroxysmal coughing. The patient’s symptoms started after an episode of violent coughing, and continued coughing contributed to the rib fractures and the diaphragmatic rupture. The patient required surgical repair for the diaphragm and fractured ribs, leading to an uneventful recovery.

Infection of Left Atrial Thrombus Associated With Mitral Stenosis*

A Case Report

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We describe the first reported case of an infected left atrial thrombus. The case of the 65-year-old male patient in this report was associated with mitral stenosis and involved Escherichia coli, and was treated successfully with surgical resection of the

References

infected thrombus. This case suggests that such infection should be considered as a possible complication of intracardiac thrombus when bacteremia is present. (CHEST 2000; 117:1201–1203)

Key words: infected thrombus; mitral stenosis; transesophageal echocardiography

Abbreviation: TEE = transesophageal echocardiography

Systemic thromboembolism is a common complication of cardiac mural thrombosis. However, infection of a ventricular mural thrombus is a very rare condition, with only occasional case reports in the literature.1–10 Furthermore, to our knowledge, infection of an atrial thrombus has not been previously described. The case reported here, associated with mitral stenosis and involving Escherichia coli, is the first report of an infected left atrial thrombus.

CASE REPORT

A 65-year-old man with no significant medical history was referred to the ICU of Kitaishikai Hospital with a spiking fever (duration, 7 days) accompanied by hypotension. He had no history of valvular disease or arrhythmia. His pulse rate was 94 beats/min and irregular, with systolic BP of 64 mm Hg. A chest examination revealed an accentuated first heart sound, an opening snap, and a diastolic rumbling murmur. The WBC count was 38,800 cells/μL (93% neutrophils), and C-reactive protein was 15.6 mg/dL. BUN was 42 mg/dL, and creatinine was 2.4 mg/dL. Urinalysis found about 100 WBCs per high-power field. An ECG showed atrial fibrillation. The chest radiograph revealed left atrial enlargement with a double right-sided heart border. Therapy was started with catecholamine, γ-globulin, and broad-spectrum antibiotics (panipenem/betamipron, 1 g/d) on a high suspicion of septic shock after three blood cultures were obtained; in 7 days, E coli was grown from all the blood cultures.

Abdominal ultrasound scans and a gallium scan disclosed no abnormalities. Systemic CT showed no abnormality except a large left atrial thrombus. Transthoracic echocardiography showed mitral valve stenosis, the giant left atrium, and a large left atrial thrombus with a mobile echo. Transesophageal echocardiography (TEE) showed the left atrial thrombus with an undulating membrane (Fig 1, left, A) and a string-like echo floating in the left atrium (Fig 1, right, B). This mobile object seemed to have a fragile stalk. Systemic embolism was a possibility. Signs of sepsis and the spiking fever continued despite treatment with gentamicin and piperacillin, which showed good antimicrobial sensitivity in vitro. Thus the patient underwent urgent surgery for a suspected infected mural thrombus.

During surgery, we observed a large thrombus covered with a white membrane enclosing purulent fluid. The string-like echo was not found to be a vegetation but rather a piece of torn membrane. There was no vegetation of the mitral valve. The infected thrombus was removed, and open mitral commissurotomy was performed. The culture of the thrombus grew E coli; on histologic examination, the thrombus demonstrated abundant neutrophils and fibrin. The patient’s postsurgical condition was uneventful.

DISCUSSION

Infection of intracardiac thrombus is very rare, with only 10 previously reported cases associated with myocar-
Tight atrium. This observation of the thrombus was similar to the description by Schofield et al. 8

TEE showed a thrombus with the unique appearance of a membrane separating it from the cavity of the left atrium. The thrombus seems to be different from that of native valve infective endocarditis. Since the frequency of mitral stenosis with thrombus is high, one might wonder why infection of an atrial thrombus is seemingly rare. The patient in our case was healthy before admission and not immunocompromised. In 8 of the 10 prior cases, organisms were found in cultures from the blood or thrombus. Interestingly, seven of these eight cases were, like the present case, due to Gram-negative bacilli (Salmonella, E. coli, Pseudomonas, Proteus, and Klebsiella). Since these organisms are rather unusual among cases of native valve infective endocarditis, the pathophysiology of infected thrombus seems to be different from that of native valve infective endocarditis.

TEE was very helpful in the diagnosis of the present case. TEE showed a thrombus with the unique appearance of a membrane separating it from the cavity of the left atrium. This observation of the thrombus was similar to the description by Schofield et al. 9

Infection of cardiac mural thrombus has a high mortality because of the difficulty of diagnosis. The patients in 6 of the previously reported 10 cases died without accurate diagnosis of the infected thrombus. 1–6,9

Thrombus is generally hypovascular tissue, so ingress of antibiotics and immune system antibodies via vasculature is thought to be poor. Thus, prompt surgical resection of the infected thrombus should be performed, followed by prolonged administration of antibiotics.

In conclusion, we present here the first case describing an infection of an atrial thrombus treated successfully with surgical resection. Such infection should be considered as a possible complication of intracardiac thrombus when bacteremia is present.

References


Echocardiographic Follow-up of Chlamydia psittaci Myocarditis*  

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Chlamydia psittaci myocarditis has infrequently reported. A case of serologically confirmed C. psittaci myocarditis with dilated left ventricle and severely impaired left ventricular function is described. Serial echocardiograms demonstrated complete recovery after therapy. An early diagnosis has important prognostic implications.

(CHEST 2000; 117:1203–1205)

Key words: Chlamydia psittaci; echocardiography; myocarditis

Abbreviations: ACE = angiotensin-converting enzyme; LV = left ventricle; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association

Psittacosis is a systemic infection caused by the obligate intracellular bacterium Chlamydia psittaci. Psittacosis is common in apparently healthy birds and domestic animals, and it is generally transmitted to man by aspiration of bird-contaminated particles. 1 In man, psittacosis usually presents as a respiratory infection giving rise to atypical pneumonia; however, rarely, severe extrapulmonary manifestations may occur. A few cases of cardiac involvement of psittacosis including myocarditis, pericarditis, and endocarditis have been reported. 2–6

We describe a 38-year-old man suffering from serologically confirmed psittacosis. He presented with atypical pneumonia and myocarditis with a strongly dilated left ventricle (LV), and severely decreased left ventricular ejection fraction (LVEF). Serial echocardiograms demonstrated complete recovery of LV function and normalization of LV dimensions after antibiotic therapy in combination with angiotensin-converting enzyme (ACE) inhibition, diuretics, and digoxin over a follow-up period of 1 year.

Case Report

A 38-year-old man was admitted to the hospital due to nonproductive cough, dyspnea on exertion, and increasing fatigue.

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CHEST / 117 / 4 / APRIL, 2000