Due to the located nature and posterior location of the effusion, the patient was taken to median sternotomy rather than attempted pericardiocectomy. At surgery, approximately 500 ml of bloody fluid was evacuated and the posterior pericardium was explored. No active areas of bleeding were noted. Of note, cultures from the pericardial fluid grew Staphylococcus epidermidis; subsequently blood cultures drawn on admission and two sets drawn postoperatively were also positive for S epidermidis. He achieved bacteriologic cure after six weeks of treatment with vancomycin and rifampin and two weeks with gentamicin.

**DISCUSSION**

Pericardial effusion after cardiac surgery is not an uncommon finding and is seen in up to 75 to 85 percent of patients.6 The incidence of effusion is highest in the first postoperative week, and early effusion progressing to tamponade is usually the result of postoperative bleeding. Late effusions are often the result of postpericardiotomy syndrome and typically have a more benign course. While this patient was therapeutically anticoagulated, no localized bleeding was noted at surgery.

The particular issue of loculated effusions is an important one in the postoperative period, and location as well as size appear to determine their course. Effusions in association with thin-walled, low-pressure atria can cause a rapidly progressive low-output state, even in the absence of a large effusion as it did in this patient. The echocardiographic correlate of tamponade, right atrial collapse, was particularly striking in this patient whose effusion was immediately adjacent to the right atrium. Similarly, the abrupt change in the chest x-ray findings, within a four-day time frame, documented the rapid accumulation of fluid which also contributed to development of tamponade physiology.

Finally, the infectious aspects of the case bear discussion. The development of tamponade in this patient led to an early diagnosis of intravascular infection. The peripheral manifestations of prosthetic valve endocarditis may not yet have been evident before antibiotic therapy was begun. Alternatively, the patient may have had surgical pericardial infection and septicemia without valve involvement. Nonetheless, treatment for early prosthetic valve endocarditis was administered as the more conservative approach.

**REFERENCES**

5. Reid CL, Rahimtoola SH, Chandraratna PA. Frequency and significance of pericardial effusion detected by two-dimensional echocardiography in infective endocarditis. Am J Cardiol 1987; 60:394-95

**Chronic Cough as the Sole Manifestation of Hodgkin's Disease**

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A patient presented with a cough of three months' duration as the sole manifestation of mediastinal Hodgkin's disease. Systematic evaluation resulted in prompt diagnosis and specific successful treatment of both the Hodgkin's disease and the cough. This case emphasizes that specific therapy based upon an accurate diagnosis almost always results in effective treatment of chronic cough.

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**MIC** = methacholine inhalational challenge; **MOPP** = combined chemotherapy with mechlorethamine, vincristine, prednisone, and procarbazine

Chronic cough is a common diagnostic and therapeutic problem. It has been shown that by using a systematic approach, one can almost always successfully identify and treat the cause of chronic cough.1-3 In nonsmokers the vast majority of cases of chronic cough is due to one or more of the following: postnasal drip; asthma; and gastroesophageal reflux;4 however, a large number of other conditions can cause chronic cough, sometimes in the absence of any other symptoms.1-4,6 We describe the first reported case, to our knowledge, of chronic cough as the sole manifestation of Hodgkin's disease.

**CASE REPORT**

A 35-year old nonsmoking man was referred to Cooper Hospital/University Medical Center for a nonproductive cough of three months' duration. Treatment with an over-the-counter cough preparation had been unsuccessful. The cough had become so severe that the patient frequently experienced near-syncope, and he was unable to carry out his usual daily activities. It occurred when he was upright and subsided when he was supine. The patient denied a history or symptoms of asthma, postnasal drip, sinussitis, gastroesophageal reflux, swallowing dysfunction, weight loss, fevers, night sweats, lymphadenopathy, or pruritus. He was taking no medications. On physical examination, he had almost constant spasms of uncontrollable coughing. He was afebrile. On examination, there was no sinus tenderness, nasal congestion, pharyngeal irritation, stridor, wheezes, crackles, hepatoplenomegaly, or lymphadenopathy.

A chest roentgenogram (Fig 1) performed five days earlier demonstrated an anterior mediastinal mass. The mass and the cough were studied simultaneously. The mediastinal mass was a potential but rare cause of cough, and we believed that we needed to consider the much commoner causes before concluding that the mass was causing the cough. While we awaited a computed tomographic (CT) scan of the thorax, the patient was treated empirically with azatadine maleate and pseudoephedrine (Trinalin) without improvement in the cough. Spirometric values and a flow-volume loop were normal, and a methacholine inhalational challenge (MIC) was negative. The CT scan confirmed the presence of an anterior mediastinal mass which impinged upon the lower trachea (Fig 2). Biopsy through a left mediastinotomy demonstrated nodular sclerosing Hodgkin's...
Disease. A staging workup was negative for disease beyond the thorax, except for possible supravacular adenopathy.

The patient was treated with a combined regimen of mechlorethamine (nitrogen mustard), vincristine, prednisone, and procarbazine (MOPP). Within two weeks of the first cycle, his cough had completely resolved. A chest roentgenogram obtained three weeks after the first cycle demonstrated nearly complete resolution of the mediastinal mass. Because of persistent thrombocytopenia following his third cycle of MOPP, chemotherapy was discontinued, and treatment was completed with radiation therapy. A chest roentgenogram taken after completion of the radiation therapy was normal. The patient remains free of symptoms and in apparent remission.

Discussion

Cough receptors are located in the ears, nose, sinuses, pharynx, larynx, trachea, bronchi, pleura, diaphragm, stomach, and pericardium.6 Afferent impulses are transmitted via the vagus, trigeminal, glossopharyngeal, or phrenic nerve to a medullary cough center.6 Efferent impulses are then transmitted via the phrenic and spinal nerves to the exhalatory muscles.6 Any disorder that affects either the cough receptors or the afferent pathways themselves is capable of producing cough.6 Chronic cough due to either intraluminal or extraluminal tracheal masses has been previously reported.6

In this case the cause of the patient's cough appeared to be direct stimulation either of tracheal cough receptors or of the afferent vagal nerve itself by the mediastinal mass. The two most common causes of chronic cough, postnasal drip and asthma,1,3 were effectively excluded, since the patient had no symptoms of postnasal drip, had no response to an empiric course of an antihistamine-decongestant combination, and had normal spirometry with a negative MIC. Compression of the trachea was evident on the CT scan of the thorax, and the cough resolved promptly with specific therapy that reduced the size of the mass. No other specific or nonspecific therapy for cough was employed during this time. Six months later, the patient remains in remission clinically and radiographically and is free of cough.

Although the chest roentgenogram was the key study in this case, we do not advocate that chest roentgenograms be routinely obtained in the initial evaluation of the nonsmoking patient with chronic cough. Roentgenographic studies will be of little value in the majority of such cases of chronic cough; postnasal drip and asthma are the most common causes,1,3 and the presence of a roentgenographic abnormality does not ipso facto prove that the abnormality is the cause of cough. Postnasal drip and asthma can usually be identified by clinical examination, empiric treatment for postnasal drip, and the results of spirometry with MIC. We would therefore argue for a sequential workup of chronic cough which addresses these common causes before roentgenograms are ordered. This approach will avoid unnecessary chest roentgenograms but will result in little delay in obtaining a roentgenogram in those cases where it will prove useful.1,3

This case is important for three reasons. First, it is the first reported case, to our knowledge, of chronic cough as the sole manifestation of Hodgkin's disease. Secondly, the case is a reminder that with cough, we too often treat the symptom and not its cause. Thirdly, this case reemphasizes that specific therapy, based on accurate diagnosis, is almost always successful in the treatment of chronic cough.

References

1. Irwin RS, Conwell WM, Pratter MR. Chronic persistent cough in the adult: the spectrum and frequency of causes and successful outcome of specific therapy. Am Rev Respir Dis 1981; 123:413-17