induced pulmonary edema, which may be caused by hypoxic-induced pulmonary venous constriction and cytokine activation. Echocardiography and/or measurements of cardiac output and intravascular filling pressures in heart failure patients during hyperbaric oxygen treatment might be informative regarding the etiology of pulmonary edema.

These patients were all treated in the single-person, monoplace oxygen-filled hyperbaric chamber (Sechrist; Anaheim, CA), necessitating the patient to be in a supine position, which might increase the risk of pulmonary edema. A difference in the incidence of pulmonary edema associated with hyperbaric oxygen therapy in supine patients vs upright patients is unknown.

Fluid overload could contribute to pulmonary edema associated with hyperbaric oxygen therapy. Death due to postoperative pulmonary edema following excess fluid retention has been reported; the authors concluded that careful attention to fluid balance was imperative. Our cases occurred in outpatients who were functional. None of these patients had recent surgery or overt manifestations of heart failure. Nevertheless, if the hyperbaric service recommends hyperbaric oxygen therapy in patients with compromised LV function, careful attention to fluid balance during their course of therapy is advised.

In case 3, it is possible that the patient’s history of radiation therapy made her disease process more complicated than just ischemic coronary disease and aortic stenosis. Radiation therapy causes tissues to become hypoxic, hypocellular, and hypoxic. This scarred fibrotic tissue might have altered her pericardium or her cardiac function. Furthermore, her aortic stenosis may have contributed to increased afterload to a marginal left ventricle. It is interesting and of concern that this patient had no symptoms compatible with congestive heart failure and had good exercise tolerance.

Acute pulmonary edema is not expected in patients treated with hyperbaric oxygen therapy, yet pulmonary edema may occur in certain patients with heart failure. Most patients treated with hyperbaric oxygen have hypoxic wounds, which often occur in patients with ischemic cardiovascular disease who may be at risk for acute pulmonary edema during hyperbaric oxygen therapy. Unfortunately, we cannot identify in whom or when acute pulmonary edema may develop. Caution is recommended in treating heart failure patients with hyperbaric oxygen.

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REFERENCES

Stress Thallium-201 Myocardial Scintigraphy in Patients With Complete Occlusion of the Left Main Coronary Artery*

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Complete occlusion (CO) of the left main coronary artery (LMCA) is a rare but often fatal condition. The diagnosis is frequently missed because the signs and symptoms are often obscure and diverse. We describe three patients with CO-LMCA who showed unusual myocardial scintigraphic findings. The patients had extensive right-to-left collateral channels and decreased uptake and washout rates at the basal anterior and anterolateral portions of the heart wall during stress thallium-201 scintigraphy. The basal anterior to anterolateral portion of the heart wall is the most distant from the collateral artery and should be the most ischemic area shown during exercise, resulting in this scintigraphic pattern. This scintigraphic finding may be useful for the noninvasive diagnosis of CO-LMCA.

(CHEST 2001; 120:1409–1412)

Key words: complete occlusion; left main coronary artery; stress thallium-201 myocardial single photon emission CT; washout rate

Abbreviations: CO = complete occlusion; EF = ejection fraction; LAD = left anterior descending artery; LCX = left circum-

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flex artery; LMCA = left main coronary artery; LV = left ventricle; ventricular; LVEDP = left ventricular end-diastolic pressure; RCA = right coronary artery; SPECT = single photon emission CT.

Complete occlusion (CO) [atherosclerotic] of the left main coronary artery (LMCA) is an extremely rare (incidence, 0.043 to approximately 0.067%) but often fatal condition. Patients with CO-LMCA are at risk for massive myocardial infarctions and sudden death. Immediate revascularization is recommended in an effort to prolong life and to alleviate symptoms in these patients.

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However, the diagnosis is still frequently missed until the patient undergoes selective coronary arteriography because the signs and symptoms can be obscure at times and the clinical manifestations are often diverse. In this report, we describe three patients with CO-LMCA who had characteristic findings on stress thallium-201 myocardial scintigraphy.

CASE REPORTS

Case 1

A 62-year-old man first noted the onset of retrosternal discomfort at the beginning of an morning walk 6 months prior to hospital admission. Subsequently, he experienced almost weekly angina at work. He had a history of hypertension, diabetes mellitus, and cigarette smoking. A physical examination revealed a BP of 166/92 mm Hg and a pulse of 66 beats/min. The resting ECG showed only nonspecific ST-T abnormalities, and the chest radiograph revealed no abnormalities. The result of a Master's two-step test was positive with a 2-mm ST-segment depression.

The patient was admitted to our hospital and was hospitalized for further evaluation in May 1987, at which time a stress 201TI myocardial single photon emission CT (SPECT) scan was performed. An exercise-induced 201TI perfusion defect was observed only in the basal anterolateral wall of the left ventricle (LV), and the defect was redistributed at rest (Fig 1, top, A). The myocardial 201TI washout rate, displayed in a bull's-eye-view map (Fig 1, top, A), was decreased in the basal anterolateral wall. Based on circumferential profile analysis using apical, midventricular, and basal short-axis images, the circumferential washout rate curve for the base was significantly decreased in the anterolateral segment (Fig 2, top, A).

At the time of cardiac catheterization, the LV end-diastolic pressure (LVEDP) was 6 mm Hg and the ejection fraction (EF) was 0.68. Left ventriculography revealed mild anterior wall hypokinesis. The LMCA was completely occluded, and the left anterior descending artery (LAD) and left circumflex artery (LCX) were easily visualized during right coronary artery (RCA) injection via extensive intercoronary collateral channels (Fig 3). The collateral pathways were mainly formed by interventricular septal branches, the distal posterior descending artery connecting around the cardiac apex to the distal LAD and the distal posterior lateral ventricular branch of the RCA. The RCA was a dominant vessel with noncritical stenoses.

The patient underwent coronary artery bypass grafting of the LAD and the LCX. Three months later, the grafts were found to be patent, and the stress 201TI myocardial SPECT scan showed no perfusion abnormality.

Case 2

A 73-year-old woman with an 8-year history of hypertension and a 3-year history of diabetes consulted a physician for the treatment of hypertension. She had smoked one pack of cigarettes per day for 35 years. She had no anginal symptoms. On physical examination, the BP was 174/94 mm Hg, the pulse was 68 beats/min, and the lung fields were clear. Chest radiography showed a normal heart size and normal pulmonary vascular markings. The resting ECG showed evidence of left ventricular...
hypertrophy, and a treadmill exercise ECG using a Bruce protocol was positive with 1.5 mm of additional ST-segment depression. However, the patient did not experience chest pain during exercise. She was referred for further evaluation and was hospitalized.

During a stress $^{201}$TI myocardial SPECT scan, an exercise-induced $^{201}$TI perfusion defect, which redistributed at rest (Fig 1, middle top, B), was observed in the basal anterior and mid-anterolateral wall of the LV. A distinct decrease in myocardial $^{201}$TI washout rate was noted in the basal anterior and mid-anterolateral wall of the LV on the bull's-eye view map (Fig 1, middle top, B) and the circumferential profile curve (Fig 2, middle bottom, C). The patient subsequently underwent cardiac catheterization. Left ventriculography revealed mild hypokinesis of the basal anterior wall. The LVEDP was 9 mm Hg, and the EF was 0.72. Coronary arteriography revealed CO-LMCA with extensive right-to-left collateral channels filling the LAD and the LCX. The dominant RCA had a noncritical narrowing. The patient underwent bypass grafting of the LAD and LCX the next month. When the patient underwent coronary angiography 3 months later, the grafts were patent, and the LAD and LCX were partially opacified during RCA injection with relatively poor collateral channels. A stress $^{201}$TI myocardial SPECT scan showed no abnormal findings.

**Case 3**

A 72-year-old man with a 1-year history of classic angina pectoris had noticed a marked reduction in exercise tolerance and an increase in the frequency and duration of angina over a 6-month period. He had a history of subendocardial infarction of the anterior wall 7 years before presentation. On examination, the BP was 140/60 mm Hg and the pulse was 61 beats/min. The physical examination was unremarkable. The resting ECG showed complete right bundle-branch block. He was hospitalized in June 1998 for further evaluation.

A stress $^{201}$TI myocardial SPECT scan showed an exercise-induced perfusion defect, which redistributed at rest (Fig 1, bottom middle, C), in the basal anterior wall of the LV. A distinct decrease in the myocardial $^{201}$TI washout rate in the basal anterior wall on the bull's-eye view map (Fig 1, bottom middle, C) and the circumferential profile curve also were noted (Fig 2, bottom middle, C). Cardiac catheterization revealed a LVEDP of...
9 mm Hg and an EF of 0.62. Left ventriculography showed mild hypokinesis of the basal anterior wall. Coronary arteriography revealed CO-LMCA with extensive right-to-left collateral channels filling the LAD and LCX. The dominant RCA had a proximal 75% stenosis. The patient underwent bypass grafting of the LAD and the LCX. The grafts were patent 3 months later, and the patient has been free of symptoms during exertion. A stress 201TI myocardial SPECT scan showed no abnormality.

**Discussion**

The patients described in this report had characteristic scintigraphic findings on stress 201TI myocardial SPECT scans. A distinct decrease in the 201TI washout rate was most prominent in the basal anterior and/or anterolateral wall, where a decreased 201TI washout rate is not usually observed in patients with ischemic heart disease. In the setting of proximal stenosis of the LAD, the most prominent redistribution and decreased washout rate are commonly found in the mid to apical region of the anterior LV wall distal to the lesion on stress 201TI myocardial SPECT scanning7,8 (Fig 1, bottom, D and Fig 2, bottom, D). In contrast, in the setting of CO-LMCA with extensive right-to-left collateral channels, the LAD and the LCX territories are perfused in a retrograde manner by collateral flow. Therefore, the basal anterior and anterolateral wall of the LV, which is closest to the LMCA and furthest from the RCA, should be the most ischemic area during exercise.9 This could explain the unique scintigraphic pattern seen in the patients with CO-LMCA. The differences in the scintigraphic patterns between patient 1 and patient 3 might be caused by the extent of the collateral channels to LCX, which were less extensive in patient 1.

In summary, we described three patients with CO-LMCA who had decreased uptake and washout rates in the basal anterior and anterolateral wall of the LV on stress 201TI myocardial SPECT scans. We suggested that this unusual characteristic scintigraphic pattern may be a specific finding for this rare condition and may be useful for the noninvasive diagnosis of CO-LMCA.

**References**


**Stenting To Reverse Left Ventricular Ischemia Due To Left Main Coronary Artery Compression in Primary Pulmonary Hypertension**

Stuart Rich, MD, FCCP; Vallerie V. McLaughlin, MD; and William O’Neill, MD

Angina is a common symptom of severe pulmonary hypertension. Although many theories for the source of this pain have been proposed, right ventricular ischemia is the one most commonly accepted as the cause. We report on two patients with primary pulmonary hypertension who had angina with normal activity or on provocation. One patient had severe left ventricular dysfunction. Both were found to have severe ostial stenosis of the left main coronary artery as a result of compression from a dilated pulmonary artery. Both patients underwent stenting of the left main coronary artery with excellent angiographic results, and complete resolution of the signs and symptoms of angina and left ventricular ischemia. Left ventricular ischemia due to compression of the left main coronary artery may be a much more common mechanism of angina and left ventricular dysfunction in patients with pulmonary hypertension than previously acknowledged. Stenting of the coronary artery can be done safely with the resolution of these symptoms.

**(CHEST 2001; 120:1412–1415)**

**Key words:** coronary artery stenting; left main coronary stenosis; primary pulmonary hypertension

**Abbreviation:** PPH = primary pulmonary hypertension

Angina, like chest pain, commonly has been associated with the development of primary pulmonary hypertension (PH).1 The etiology of the chest pain has been debated, with theories ranging from painful dilatation of

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