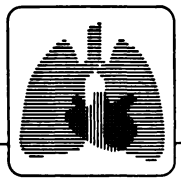

selected reports

Mediastinal Lymph Node Enlargement as a Result of Mitral Valve Stenosis*

Marjolein Drent, M.D.; Jan-Philip Gelissen, M.D.; Carl A. P. L. Ascoop, M.D., F.C.C.P.; Sjoerd Sc. Wagenaar, M.D.; and Jules M. M. van den Bosch, M.D., F.C.C.P.

Two patients are described with severe MVS, pulmonary venous hypertension and enlarged mediastinal, pulmonary and hilar lymph nodes. These enlargements were diagnosed on a preoperative chest CT. After MV replacement these enlarged lymph nodes nearly all resolved. The lymphadenopathy should be considered to be secondary to MVS with pulmonary venous hypertension.

(Chest 1992; 102:1269-71)

LA = left atrium; MV = mitral valve; MVS = MV stenosis

*From the Departments of Pulmonary Diseases (Drs. Drent and van den Bosch), Radiology (Dr. Gelissen), Cardiology (Dr. Ascoop) and Pathology (Dr. Wagenaar), St. Antonius Hospital, Nieuwegein, the Netherlands.

This project was supported by a grant from Glaxo BV, the Netherlands.

Reprint requests: Dr. van den Bosch, St. Antonius Hospital, Koekoekslaan 1, Nieuwegein, the Netherlands 3435CM

Mediastinal lymph nodes are one of the most common causes of abnormal mediastinal masses. Two cases of enlarged mediastinal lymph nodes, MVS and pulmonary venous hypertension are described and a literature survey is given.

Case Reports

Case 1

A 45-year-old woman was admitted to the hospital for MV replacement because of severe MVS. The chest radiograph showed a dilated LA and pleural effusion at both sites. A preoperative chest CT scan showed serial enlarged hilar and mediastinal lymph nodes on both sides. Mediastinoscopy showed multiple lymph nodes. An enlarged lymph node was excised, without signs of malignancy or infection. All clinical details are summarized in Table 1. After surgical treatment, MV replacement by a Sorin prosthesis, there was a complete resolution of the patient's symptoms.

Case 2

A 49-year-old woman was admitted to the hospital for investigation of mediastinal lymphadenopathy. She had a clear pulmonary history. At the age of 10 years she had rheumatic fever. The chest radiograph showed a dilated LA and enlarged mediastinum, with some fluid in the interlobar area on the right side. A chest CT scan showed enlarged lymph nodes in the superior mediastinum, a large hilus on the right side and some infiltration of the right, middle and lower pulmonary lobes (Fig 1 and 2). Heart catheterization revealed a MVS with MV regurgitation and pulmonary hypertension.

Surgical intervention by replacing the MV with a Sorin prosthesis was necessary because of the severe MVS. A mediastinal lymph node (2R, according to the ATS lymph node mapping scheme) was removed with focal dilated intranodular lymph vessels, but no signs of malignancy (Fig 3, Table 1).[1-3]

Table 1—*Clinical and Diagnostic Results of Cases 1 and 2*

Clinical Data	Case 1	Case 2
Age (yr)	45	49
Sex	F	F
Clinical record	Fatigue, cough, no fever, progressive exertional dyspnea, hoarseness	Strangling sensation in the chest and dyspnea provoked by exercise, no cough, no fever
Clinical history	No abnormalities	No abnormalities
Pulmonary history	No asthma, no repeating pulmonary infections, no allergy, no tuberculosis or animal contact	No asthma, no repeating pulmonary infections, no allergy, no tuberculosis or animal contact
Smoking	10-15 cigarettes a day	20 cigarettes a day
Medication	Lorazepam, 1 mg twice a day	Temazepam, 10 mg once a day
Physical examination	No abnormalities	No abnormalities
Auscultation	Loud first heart sound, a tight mitral opening snap followed by a roulement	A holosystolic souffle II/IV, a roulement
Laboratory data	No abnormalities	No abnormalities
Serology	No abnormalities	No abnormalities
Sputum/pleural fluid	Culture; ZN and cytology: negative	Culture; ZN and cytology: negative
ECG	Signs of RA and LA enlargement	No abnormalities
Echocardiography	MVS with calcification in the MV, pulmonary hypertension	MVS, calcification in the MV, pulmonary hypertension
Wedge pressure	22 mm Hg (11 mm Hg*)	21 mm Hg (8 mm Hg*)
Pulmonary artery pressure	Systolic: 50 mm Hg (40 mm Hg*) Diastolic: 20 mm Hg (10 mm Hg*)	Systolic: 57 mm Hg (20 mm Hg*) Diastolic: 20 mm Hg (8 mm Hg*)
MV area	0.74 sq cm	0.91 sq cm
Gradient MV (exertional, end diastolic)	36.7 mm Hg	30 mm Hg
PA lymph node	reactive hyperplasia; focal congestion (2,5 × 1,5cm)	reactive hyperplasia; focal congestion; 2R[1-3] (2,4 × 1,7cm)

*Value five days after surgery.

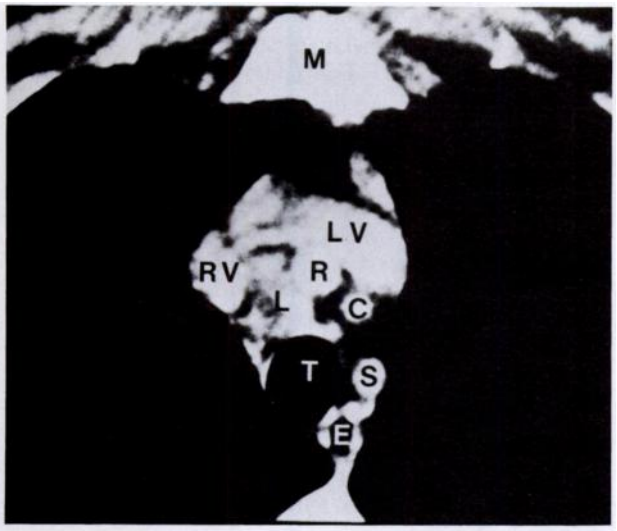

FIGURE 1. Chest CT scan, case 2, before MV replacement. *M* = manubrium; *LV* = left brachiocephalic vein; *RV* = right brachiocephalic vein; *L* = lymph node; *R* = right brachiocephalic artery; *C* = left carotid artery; *S* = left subclavian artery; *E* = esophagus; *T* = trachea.

DISCUSSION

Acquired heart diseases leading to pulmonary vascular disease have a basic physiologic abnormality: pulmonary venous hypertension. The list includes stenosis of the pulmonary veins, cor triatriatum, MVS (best studied) and left ventricular failure of any etiology.[4-6]

Pulmonary venous hypertension affects the pulmonary circulation, the lymph drainage and the bronchial circulation. At the time MVS becomes progressively severe, changes that will occur are pulmonary venous hypertension, at first passive and then reactive, a reduction of flow across the MV, right ventricular hypertrophy and failure as the pulmonary arterial pressure rises to high levels. The pulmonary vascular resistance only increases when the MV orifice approaches 1.0 sq cm and the pulmonary wedge pressure is 25 mm Hg.[5]

The pulmonary vascular responses to MVS are a modest degree of morphologic response of pulmonary arterioles and muscular arteries. These responses are not to be that severe

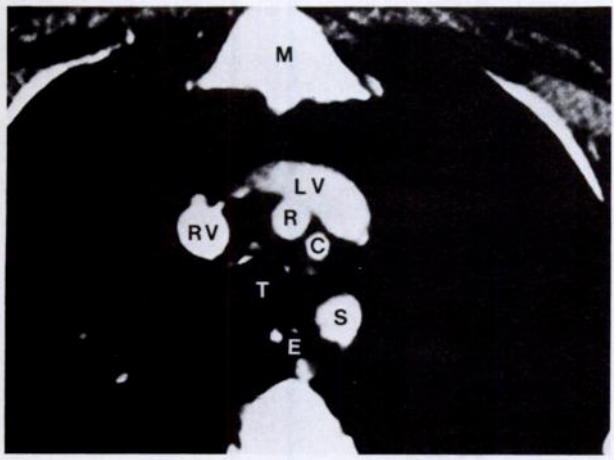

FIGURE 2. Chest CT scan, case 2, after MV replacement. *M* = manubrium; *LV* = left brachiocephalic vein; *RV* = right brachiocephalic vein; *R* = right brachiocephalic artery; *C* = left carotid artery; *T* = trachea; *E* = esophagus; *S* = left subclavian artery.

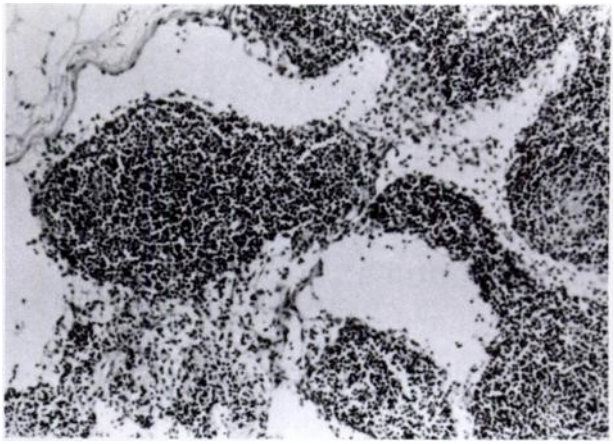

FIGURE 3. PA case 2. Reactive hyperplasia; focal congestion; no signs of malignancy (2,4 × 1,7-cm specimen).

that they will become irreversible and self-perpetuating after relief by MVS surgery. The increased transmural pressure is the so-called reactive pulmonary vasoconstriction in chronic MVS.[4,5] Lymphatic distention has been seen as pulmonary edema develops.[5,6] Enlargement of hilar and mediastinal lymph nodes or mediastinal lymphadenopathies can be caused by various disorders (Table 2).[7-14]

The pulmonary lymphatic system in humans is organized into two main sets of vessels: the superficial or pleural network and the deep or peribronchovascular network.[11,12] These two systems anastomose in the pleura or at the hilus.[11,12] The pulmonary lymphatic system plays a major role in the fluid circulation of the lung.[6,12,15,16]

The overloading of pulmonary lymph drainage, caused by the presence of pulmonary venous hypertension, is the additional factor in lymphadenopathy.[15-18] In a text by Dexter,[5] Kerley described A and B lines attributed to lymphatic vessels. Similar lines were soon recognized in MVS and lymphangitis carcinomatosa.

Dilated interlobar and pleural lymphatics are confined

Table 2—*Mediastinal Lymphadenopathies*

Malignant lymphomas
Hodgkin's disease
Non-Hodgkin's lymphoma
Lymphangioma
Metastatic and other neoplasms
Granulomatous lymphadenopathies and lymphadenitis
Tuberculosis
Fungal infections: histoplasmosis, coccidiodomycosis, others
Sarcoidosis
Silicosis
Wegener's granulomatosis
Others
Angiofollicular hyperplasia (Castleman's disease)
Angioimmunoblastic lymphadenopathy
Pulmonary lymphangiectasis
Primary: congenital
Secondary: pulmonary venous hypertension or obstruction
Lupus erythematosus, rheumatic arthritis
Infectious mononucleosis
Drug-induced lymphadenopathy
Reactive lymph node hyperplasia
MVS with pulmonary venous hypertension

Table 3—*Results of Other Investigators*

Levin[16] (1955): 63 cases of MVS
Enlarged septal lymphatics in 39 cases
Positive correlation between the degree of lymphatic dilatation and PA and wedge pressures
No correlation between lymphatic dilatation and pulmonary hypertension of other than MVS etiology
After commissurotomy decreasing and disappearing lymphectasia
Heath and Hicken[17] (1960): 20 cases of MVS
"Pulmonary lymphangiectasis"
Distended lymphatics and nodes around pulmonary arteries
Six of seven patients, wedge-pressure >/30 mm Hg
Tandon and Kasturi[18] (1975): 100 cases of MVS
Dilated lymphatics in the pleura and interlobar septa: 40 cases
Hypertrophied muscular wall of the lymphatics: a few cases

almost entirely to persons with pulmonary hypertension secondary to MVS.[5,6,19] This is probably a reflection of sustained elevated LA pressure with consequent pulmonary and bronchiolar venous and capillary hypertension. With high pulmonary and bronchiolar venous and capillary pressures the lymphatics may well serve as a device to carry the transudate away, which could have otherwise caused pulmonary alveolar edema. Such edema is not often seen in MVS.[16,17]

Dilatation of lymph canals can be a result of pulmonary venous hypertension caused by MVS (Table 3). We have not found any earlier reports about lymph node congestion and MVS; it was mentioned only once in relation to a frequent symptom of MVS hoarseness. This should be caused by compression of the recurrens nerve by the pulmonary artery and enlarged lymph nodes.

The two cases we reported had a control CT scan a year and a half after surgical intervention. As mentioned, a chest CT scan showed marked enlarged mediastinal lymph nodes in both patients. The short axis of the lymph node stations 2L and 2R (ATS lymph node mapping scheme) were enlarged in the first patient, which means the short axis measured over 1.0 cm in the transverse plane.[1,2] The second patient showed enlarged nodes in the 2R and 4R location, which measured, respectively, 2.0 and 1.8 within the limit. In both patients, all lymph nodes resolved or became within the limit after the hemodynamic response by MV replacement.

In conclusion, mediastinal lymphadenopathy should be considered to be secondary to MVS with pulmonary venous hypertension if a malignancy or infection is excluded or less probable. Enlargement of the mediastinal lymph nodes as a result of MVS is reversible as are pulmonary hypertension and pulmonary function disturbance.[19-21]

References

1 Naruke T, Suemasu K, Ischikawa S. Lymph node mapping and curability at various levels of metastasis in resected lung cancer. J Thorac Cardiovasc Surg 1978; 76:832-39

2 Glazer GM, Gross BH, Quint LE, Francis IR, Bookstein FL, Orringer MB. Normal mediastinal lymph nodes: number and size according to American Thoracic Society mapping. AJR 1985; 144:261-65

3 Legge DA, Miller W, Ludwig J. Pulmonary findings associated with mitral stenosis. Chest 1970; 58:403-04

4 Wagenvoort CA, Denolin H. Pulmonary venous hypertension—mechanisms and consequences. In: Pulmonary circulation—advances and controversies. Amsterdam: Elsevier Science Publishers BV, 1989:131-47

5 Dexter L. Pulmonary vascular disease in acquired heart disease. In: Pulmonary vascular diseases. New York: Marcel Dekker, 1979:427-89

6 Rabin ER, Mayer EC. Cardiopulmonary effects of pulmonary venous hypertension with special reference to pulmonary lymphatic flow. Circ Res 1960; 8:324-35

7 Marchevsky AM, Kaneko M. Surgical pathology of the mediastinum. New York: Raven Press, 1984:1-16,173-216

8 Trapnell DH. The peripheral lymphatics of the lung. Br J Radiol 1963; 36:660-72

9 Bergin C, Castellino RA. Mediastinal lymph node enlargement on CT scans in patients with usual interstitial pneumonitis. AJR 1990; 154:251-54

10 Blank N, Castellino RA. Mediastinal lymphadenopathy. Semin Roentgenol 1977; 12:215-46

11 Tobin CE. Human pulmonic lymphatics: an anatomic study. Anat Rec 1957; 127:611-33

12 Courtice FC, Simmonds WJ. Physiological significance of lymph drainage of the serous cavities and lungs. Am Physiol Soc 1954; 34:419-48

13 Sill V. The lung in heart diseases. Pneumonology 1990; 44:121-26

14 Wagenaar SjSc, Swierenga J, Wagenvoort CA. Late presentation of primary pulmonary lymphangiectasis. Thorax 1978; 33:791-95

15 Ryan CJ, Rodgers RF, Unni KK, Hepper NGG. The outcome of patients with pleural effusion of intermediate cause at thoracotomy. May Clin Proc 1981; 56:145-49

16 Levin B. On the recognition and significance of pleural lymphatic dilatation. Am Heart J 1955; 49:521-37

17 Heath D, Hicken P. The relation between left atrial hypertension and lymphatic distension in lung biopsies. Thorax 1960; 15:54-58

18 Tandon HD, Kasturi J. Pulmonary vascular changes associated with isolated mitral stenosis in India. Br Heart J 1975; 37:26-36

19 Cross CE, Shaver JA, Wilson RJ, Robin ED. Mitral stenosis and pulmonary fibrosis: special reference to pulmonary edema and lung lymphatic function. Arch Intern Med 1970; 125:248-54

20 Rolla G, Bucca C, Caria E, Scappaticci E, Baldi S. Bronchial responsiveness in patients with mitral valve disease. Eur Respir J 1990; 3:127-31

21 Nishimura Y, Maeda H, Yokoyama M, Fukuzaki H. Bronchial hyperreactivity in patients with mitral valve disease. Chest 1990; 98:1085-90

Aortico-Left Atrial Fistula in Aortic Valve Endocarditis*

Ramsey Behnam, M.D.

Aortic root abscess is a well-known complication of aortic valve endocarditis. This report describes the two-dimensional echocardiographic findings in a patient with aortic valve endocarditis whose course was complicated by a posterior aortic root abscess which ruptured into the left atrium creating an aortico-left atrial fistula, which, to our

*From the Division of Cardiovascular Medicine, Department of Internal Medicine, University of California Davis Medical Center, Davis.