The Angiocardiographic Diagnosis of Left Atrial Thrombosis

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With the advent of modern cardiac surgery the clinical diagnosis of intramural thrombi, ball valve thrombi and myxomatous and other tumors in the left atrium has assumed great importance. It is shown here that a diagnosis of thrombosis within the left atrium is possible by angiocardiography. The angiocardiographic features of mural and ball valve thrombi of the left atrium and of left auricular thrombosis are described.

THROMBI in the left atrium have been definitively diagnosed during life, in the absence of cardiac surgery, only in those very rare instances when they were sufficiently calcified to be visible on conventional roentgenograms.\(^1\)\(^2\) We report herein for the first time, so far as we are aware, their visualization by angiocardiography. We found five instances in our first 57 studies, an incidence of 8.8 per cent. This incidence may be compared to that found at autopsy in patients with mitral stenosis, which is variously reported to be between 8 and 15 per cent\(^3\)\(^4\) (excluding those thrombi localized to the left auricle). Considering that our patients are obviously in an earlier stage of the rheumatic disease than those that come to autopsy and the occasional confusion of agonal or postmortem clots with ante-mortem thrombi, we believe that our angiocardiographic incidence of intra-atrial thrombi is close to the actual incidence of such thrombi in our series of patients.

Of the 5, 2 were verified at operation and one at autopsy. Surgery was not performed in one patient, primarily because of our angiocardiographic findings. The last had no preoperative angiographic evidence. A thrombus, which was found but not removed at surgery, was demonstrated later by angiocardiography.

CASE REPORTS

Case 1. E. W., a 49 year old woman, entered the Episcopal Hospital on March 4, 1955, because of dyspnea. She had chokes at 9 and a right hemiparesis since the age of 15, when a cerebral embolus occurred 12 days after delivery of her first pregnancy. Since her tenth and last pregnancy at 36, she has had intermittent dyspnea and weakness.

Examination revealed slight enlargement of the heart, atrial fibrillation, accentuated pulmonic second sound and a faint apical diastolic rumble. The jugular veins were slightly distended. The liver was slightly enlarged.

Roentgenographic study showed a slightly enlarged cardiac silhouette. The pulmonary segment was convex. The right ventricle and left atrium were enlarged. The pulmonary vascular markings were increased. An electrocardiogram showed atrial fibrillation, rightward deviation of QRS, and nonspecific ST-T changes.

An angiogram was done on March 14, 1955. The superior vena cava time was 2.8 seconds, the right heart time 20.3, the pulmonary artery time 18.9, the left atrial time 13.3 and the intracardiac circulation time 9.8. The right atrium, right ventricle and pulmonary artery were enlarged. The left atrial volume was 303 ml. The left auricle was not opacified and was irregularly marginated from the opacified left atrium. Large filling defects were observed in the opacified left atrium, on the right, superiorly and inferiorly, and also posteriorly (fig. 1). The left ventricle was opacified to a lesser degree than the left atrium.

Mitral valvotomy was done on March 28, 1955, by Dr. Thomas J. E. O’Neill. The left auricle was enlarged and obliterated by organized thrombi. A large mural thrombus was felt along the posterior wall of the left atrium and could not be removed.

The angiocardiographic study was repeated 2 weeks after surgery and the filling defects in the left atrium were again observed.

Case 2. M. G., a 40 year old woman, entered Temple University Hospital on Sept. 12, 1955, because of progressive dyspnea and weakness.

Examination revealed moderate cardiomegaly, atrial fibrillation, accentuated pulmonic second sound and grade 2 systolic and moderate rumbling diastolic apical murmurs. Other than slight distention of the jugular veins, there were no signs of heart failure.

Roentgenographic study disclosed a moderate inc-
crease in size of the cardiac silhouette. The pulmonary segment was convex. The left atrium and right ventricle were enlarged. The pulmonary vascular markings were increased. An electrocardiogram showed atrial fibrillation, rightward deviation of QRS and nonspecific ST-T changes.

Cardiac catheterization on Sept. 19, 1955, (done by Dr. George E. Mark, Jr.), revealed a pressure in the pulmonary artery of 60/26 mm. Hg and, in the right ventricle, 64/5 mm. Hg. The cardiac output was 3.5 L./min.

Angiocardiography immediately afterward showed a prolongation of all circulation times. The superior vena cava time was 7.0 seconds, the

Fig. 1. Case 1. Simultaneous angiocardiograms in the anterior and left lateral projections, 11.2 seconds from initial opacification of the right atrium. The region of the left auricle is not opacified. There is irregular margination of the opacified left atrium at its auricular junction. In the anterior projection, note the filling defects in the opacified left atrium on the right, superiorly and inferiorly. In the lateral projection, there is sparse opacification of the left atrium superiorly and a filling defect along the posterior wall immediately above the region of the mitral valve.
right heart time 19.6, the pulmonary artery time 16.8, the left atrial time 17.7 and the intracardiac circulation time 18.2. The right atrium and ventricle, pulmonary artery and its branches were enlarged. The left atrial volume was 621 ml. Neither the left auricle nor the left ventricle was visualized. The border between the left auricle and atrium was irregularly marginated. There were large irregular filling defects protruding from the posterior wall of the left atrium in the lateral projection only (fig. 2).

Mitral valvotomy was done on Sept. 27, 1955, by Dr. George Rosemond. The left auricle was obliterated by organized thrombi. A clot was encountered by the finger entering the left atrium. Some of the clot spurted out and the remainder was flushed out. Following mitral valvotomy, it was thought that the left atrium was free of thrombus. Immediately after surgery, a saddle embolus was recognized and removed.

Two weeks later, angiocardiography again showed filling defects in the left atrium.

Case 3. A. F., a 28 year old woman with rheumatic heart disease and multivalvular involvement, entered Temple University Hospital on June 18, 1955, because of fever and intractable heart failure precipitated by a septic pulmonary infarct with abscess formation. During hospitalization, she had transient atrial fibrillation and prolonged unconsciousness due to cerebral embolism.

Roentgenographic study revealed marked cardiomegaly and a very prominent pulmonary artery. The right ventricle and left atrium were enlarged. An electrocardiogram showed sinus rhythm, large broad P waves and right ventricular hypertrophy.

An angiocardiogram was done on Sept. 7, 1955, following subsidence of fever and restoration of consciousness. The superior vena cava time was 16.8 seconds, the right heart time 56.1, the pulmonary artery time 52.6, the left atrial time 42.1 and the intracardiac circulation time 26.8. There was marked enlargement of the superior vena cava, right atrium and ventricle and pulmonary artery. The left atrial volume was 420 ml. A round filling defect was seen in the anterior and superior portion of the left atrium in the lateral projection only (fig. 3).

Cardiac surgery was not done because of bacterial endocarditis. Two months later, she was readmitted to the hospital, dyspneic, cyanotic and pulseless. She died in eight hours.

At autopsy, an organized thrombus, the size of a golf ball, was found free in the left atrium (fig. 4). There was multivalvular involvement. Vegetations were present along the mitral valve.

Case 4. M. P., a 62 year old woman, was admitted to the Episcopal Hospital on Jan. 18, 1955, because of sudden left hemiplegia. She had rheumatic fever at 30, angina pectoris since the age of 55, and, at 60, a saddle embolus, confirmed by aortography.

The blood pressure was 180/100 mm Hg. The heart was not enlarged. The pulmonic second sound was accentuated. Atrial fibrillation was present. There were no signs of failure.

The hemiplegia disappeared immediately following right stellate ganglion block.

![Fig. 2. Case 2. Angiocardiogram in the left lateral projection, 11.2 seconds from initial opacification of the right atrium. The left auricle is not opacified. There is irregular margination of the opacified left atrium at its auricular junction. There are filling defects along the posterior wall of the left atrium.](http://circ.ahajournals.org/)

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Fig. 3. Case 3. Angiocardiogram in the left lateral projection, 9.8 seconds from initial opacification of the right atrium. The left auricle is not seen and the auricular border of the opacified left atrium is smooth. Note the relatively round filling defect in the anterior, superior portion of the left atrium.

Fig. 4. The heart of case 3 at autopsy. Note the ball valve thrombus in the left atrium and the vegetations in the region of the mitral valve.

Roentgenographic study showed slight left atrial enlargement and prominence of the left second and third segments of the cardiac silhouette. An electrocardiogram showed atrial fibrillation and nonspecific ST-T changes.

An angiocardiogram was done on Feb. 1, 1955. The superior vena cava time was 5.6 seconds, the right heart time 20.3, the pulmonary artery time 18.9, the left atrial time 11.2 and the intracardiac circulation time 9.8. The left atrial volume was 303 ml. Filling defects were observed along the posterior wall of the left atrium, centrally and at the entrance of the right inferior pulmonary vein into the left atrium (fig. 5). The left auricle was not opacified and was irregularly marginated at its junction with the left atrium.

Cardiac surgery was considered unwise because of age, coronary artery disease and angiocardiographic findings.

Case 5. R. D., a 28 year old man, had polyarthritis and chorea at 11 and a cerebral embolus with right hemiparesis and transient atrial fibrillation at 25. Two years later, he entered the Episcopal Hospital for cardiac surgery.

Examination revealed right hemiparesis, slight cardiac enlargement, sinus rhythm, accentuated pulmonic second sound, pulmonary diastolic murmur and grade 2 systolic and moderate rumbling diastolic murmurs at the apex.

Roentgenographic study disclosed a slight increase in size of the cardiac silhouette with enlargement of the left atrium and right ventricle. The pulmonary artery segment was convex and the left third segment prominent. There was calcification of the mitral valve and of the posterior wall of the left atrium. An electrocardiogram showed findings consistent with left atrial disease and right ventricular hypertrophy.
Mitral valvotomy was done on Sept. 17, 1953, by Dr. Thomas J. E. O'Neill. The left auricle was thrombosed. Much of the superior portion of the left atrium was thrombosed and calcified. The thrombi could not be removed. There was calcification of the posterior wall of the left atrium and of the mitral valve.

Angiocardiography was done on Dec. 3, 1954, following recovery from a bout of heart failure. The superior vena cava time was 3.5 seconds, the right heart time 7.7, the pulmonary artery time 8.4, the left atrial time 12.6 and the intracardiac circulation time 8.4. The left atrial volume was 324 ml. The left auricle was not opacified. There was irregular margination of the left atrium superiorly. In the lateral projection, there were also large irregular filling defects in the left atrium posteriorly and superiorly (fig. 6).

**Fig. 5.** Case 4. Simultaneous angiocardiograms in the anterior and left lateral projections, 14.0 seconds from initial opacification of the right atrium. The left auricle is sparsely opacified. The junction of the opacified left atrium and the auricle is irregular. Note the large filling defects in the left atrium in each projection.
Discusssion

A retrospective diagnosis of thrombus within the left atrium is entertained when systemic embolization occurs in an individual with mitral stenosis. But one cannot be certain that the embolus actually arose from the left atrium or that a residual thrombus is present. Calcification of the posterior wall of the left atrium is sometimes associated with an underlying thrombus, but thrombi are frequently present in the absence of such calcification. Prominence of the third left segment of the cardiac silhuette in the anterior projection may also suggest a thrombus, but this finding is frequently present without a thrombus and, conversely, a thrombus is frequently present without this sign. Only calcification within the left atrium is diagnostic and this is very rare.

It has been stated that mural thrombi do not produce defects in the left atrium. On the contrary, the left atrium is particularly suitable for angiocardiographic visualization of thrombi.

Opacification of the left atrium in mitral stenosis is intense, homogeneous and persists for longer than normal. Any mass attached to the walls of the left atrium is easily visible, provided overlapping structures are eliminated and the projection is adequate. We have found the posterior-anterior and left lateral projections best for avoiding overlapping of opacified structures. The simultaneous biplane angiocardiographic apparatus of Chamberlain, which we use, permits not only two projections at right angles but also stereoscopic pairs that aid in the differentiation of extracardiac from intracardiac densities. Of greatest importance, is the projection used. In 3 of our 5 cases, filling defects were not seen in the anterior projection but were clearly visualized in the left lateral projection. The left lateral projection is best because only in this projection is the extreme posterior wall of the left atrium visible. It is from this wall that the left atrial mural thrombus arises almost always. The key to the reason for the predilection for this localization of implantation of a left atrial thrombus is present in MacCallum's classic paper of 1924, and cannot be better expressed than in his own words:

We have found several times a marked thickening of the wall of the left auricle above the base of the more posterior leaflet of the mitral valve. This roughened patch resembles in some degree the vegetations so commonly found there in cases of endo-
carditis produced by the streptococcus viridans, but is evidently more largely produced by a new growth of tissue and to a less extent by the deposition of rough fibrin on the surface. Sometimes the surface, although corrugated, seems not to be roughened by any thrombus mass. . . . In most instances the innermost layer of the auricular wall assumes a hyaline appearance over this whole area and merges gradually into a dense film of fibrin which is exposed to the blood of the auricle.

The cause of the localization of MacCallum’s patch is unknown. It may be related to mitral regurgitation, which is so often an integral part of the initial attack of rheumatic carditis, and which is present long before stenosis of the mitral valve sets in. The determining causes for thrombus formation may be the geography of the patch itself, the duration of the rheumatic state and the presence of atrial fibrillation which further slows left atrial circulation time and immobilizes effective atrial contractions. Other explanations have been suggested.3-6

Although the left lateral projection is best for visualizing these thrombi, additional projections may be of value, for they may provide supplemental data of the extent of the thrombus.

The diagnosis of thrombus of the left auricle poses a more difficult problem because the left auricle is occasionally not opacified even when it contains no thrombus. Moreover, the opacified left auricle may be obscured in the anterior projection by overlying structures, particularly pulmonary vessels; in the lateral projection it is superimposed upon the anterior superior portion of the left atrium. For the present, therefore, we believe that a diagnosis of left auricular thrombosis should be made only when there is irregular margination of the junction of the auricle with the atrium, together with sparse or no opacification of the auricle. These findings form the basis for our own correct diagnosis of left auricular thrombosis. In our series, however, all were associated with left atrial thrombosis. Read and his associates3 demonstrated in dogs the angiocardiographic findings of filling defects in the left atrium after invagination of the left auricle.

Radiographically, left atrial thrombi may be confused with tumors. The commonest left atrial tumor is a myxoma.14 Three left atrial myxomata have been recognized during life by angiocardiography.9 These tumors are usually of such a size as to produce large filling defects in the left atrium. They are not attached to the posterior wall of the left atrium and produce no filling defects in this region. They are usually attached to the atrial septal wall in the region of the fossa ovalis and this attachment may be visualized.9 With these points in mind, together with the history and physical findings, the differential diagnosis between myxoma and thrombus appears to be possible.

**Summary**

1. A diagnosis of thrombosis within the left atrium is possible by angiocardiography.
2. The angiocardiographic diagnosis of a mural thrombus within the left atrium is based upon the finding of a filling defect and irregular margination of the posterior wall of the left atrium best visualized in the left lateral projection.
3. A ball valve thrombus is suggested by the finding of a filling defect within the left atrium unattached to the left atrial wall and small compared to a myxoma.
4. Left auricular thrombosis is suggested by the finding of irregular margination of the junction of the left auricle with the left atrium and sparse or no visualization of the left auricle.

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**Summario in Interlingua**

1. Le diagnoso de thrombose sinistro-atral per medios angiocardiographic es possibile.
2. Le diagnoso angiocardiographic de un thrombo parietal intra le atrio sinistre es indicato post le constatation de un defecto plenatorii e de un margination irregular del pariete posterior sinistro-atral que se visualiza le melio in un projection sinistro-lateral.
3. Le diagnoso angiocardiographic de un
globular thrombo valvular es indicate post le constatation de un defecto plenatorì intra le atrio sinistre, non attachate al pariete sinistro-atrial et de parve dimensiones in comparation con un myxoma.

4. Le diagnose de thrombose sinistro-auricular es indicate post le constatation de margination irregular del junction del auriculo sinistre con le atrio sinistre e reducece o absente visualisation del auriculo sinistre.

REFERENCES


Thyroid is administered in large quantities, in most cases unnecessarily, to patients all over the world. In most cases no great harm results from such medication. Because atrial fibrillation is seldom noted after prolonged administration of large doses of thyroid, the authors report a case in which a patient with normal size heart was noted to have atrial fibrillation and a grade 2 systolic murmur at the apex, together with pitting edema of both legs after taking 3 grains of thyroid daily for 6 years. With omission of thyroid substance, edema diminished progressively and disappeared within 2 weeks. The systolic murmur disappeared in a month's time and previously noted atrial fibrillation was replaced by normal sinus rhythm. Only 3 cases of atrial fibrillation and one case of atrial flutter have been reported arising after thyroid administration to the euthyroid subject; each reverted to regular sinus rhythm following cessation of the use of thyroid.

Kitchell
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