Bacterial Endocarditis and Mural Thrombi in the Heart

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The infrequent occurrence of mural thrombi in the chambers of the heart is noted. The relevant literature is reviewed. The findings in eighty-two cases of bacterial endocarditis superimposed on rheumatic heart disease and in 47 examples of bacterial endocarditis without underlying rheumatic valvulitis are reported. These findings may reflect the infrequency of auricular fibrillation and of severe mitral stenosis in cases of bacterial endocarditis. The results further substantiate the origin of emboli from the valvular vegetations, bacterial or nonbacterial, rather than from the cardiac chambers, and this correlates with the bland character of most of the infarcts.

EMBOLIC phenomena occur with such frequency in the course of bacterial endocarditis as to constitute a cardinal diagnostic feature of this disease. However, it has been noted that the resultant infarcts are less often septic than would be expected if the emboli originated from the infected valvular vegetations. A previous study of this apparent paradox showed that it is the nonbacterial thrombotic vegetation that is a frequent source of these emboli in patients with rheumatic heart disease with or without superimposed bacterial endocarditis. An incidental feature of this analysis, further substantiating the above findings, was the relative rarity of mural thrombi as the origin of such bland emboli in the cases of bacterial endocarditis.

Another report also indicated the relative infrequency of severe mitral stenosis with bacterial endocarditis as well as the greater rarity of auricular fibrillation in patients with bacterial endocarditis. The role of each of these factors in the formation of mural thrombi in the left auricle has been discussed previously.

A perusal of the recent literature discloses few references dealing specifically with this subject. Garvin in a study of mural thrombi in the heart found only 1 instance of mural thrombus in 30 cases of subacute bacterial endocarditis (3.3 per cent) and none in 13 cases of acute bacterial endocarditis. He concluded that this complication was distinctly uncommon in these diseases but offered no explanation. The standard textbooks do not mention this association.

**Material and Methods**

In order to study this problem with a large series of uniformly examined cases, 8676 consecutive autopsies performed at the Queens General Hospital from 1936 to 1950 inclusive were reviewed. The hearts of 509 (5.9 per cent) of the cases thus studied were classified as showing rheumatic heart disease since they fulfilled the following criteria: (1) fusion and retraction of the mitral or tricuspid valves; (2) fusion and shortening of the chordae tendineae; (3) noncalcific fusion of the aortic valve; (4) any fusion, including calcific changes, of the aortic valve when it was associated with definite rheumatic mitral valve involvement; (5) active rheumatic heart disease with Aschoff bodies or characteristic verrucae; (6) bacterial endocarditis of the mitral or aortic valves with some evident previous distortion, but the exact estimation of the severity of rheumatic damage being difficult.

Cases showing the following changes were excluded from this study: (1) slight mitral changes (for example, tongue-like extensions of the valve cusps), or distortion which did not fulfill the criteria stated above (points 1 and 2); (2) slight aortic stenosis with or without slight mitral involvement; (3) calcific aortic stenosis with or without slight mitral changes; (4) calcification of the valve rings alone. Thus 103 cases of calcific aortic stenosis and 72 cases of doubtful origin were excluded from this study.

In the group of rheumatic hearts, bacterial endocarditis was present in 82 (16.1 per cent); in five the endocarditis was healed. These patients were compared with 47 patients with bacterial endocardidi-
tiis in whom the hearts did not meet the criteria for rheumatic heart disease described above. In each case, the occurrence of mural thrombi in the chambers of the heart, the presence of auricular fibrillation and the severity of the mitral stenosis were noted. When the cardiac rhythm was abnormal, the time of onset relative to the beginning of the valvular infection was ascertained.

The present paper deals with the relationship of mural thrombi to bacterial endocarditis.

**Results**

Mural thrombi occurred in the cardiac chambers in 8 of the 82 cases (9.3 per cent) of bacterial endocarditis superimposed on rheumatic heart disease. Four of these had regular rhythm; two had auricular fibrillation terminally. Only two patients had preceding chronic auricular fibrillation and in one of these the endocarditis was acute. Mural thrombi were noted in 2 of the 47 cases of bacterial endocarditis which did not fulfill the criteria for underlying rheumatic heart disease. Both of these had acute terminal endocarditis, one with regular rhythm and one with preceding auricular fibrillation.

In contrast to these findings, mural thrombi were found in 137 (27 per cent) of the total 509 cases of rheumatic heart disease. One hundred twenty-nine of these cases (30 per cent) occurred in the 427 cases of rheumatic heart disease without bacterial endocarditis.

Of the 72 cases with valvular lesions of doubtful origin, thrombi were present in the heart in seven cases. Five of these were associated with myocardial infarction; in the other two cases the thrombus was in the right auricle or right appendage. The rhythm was regular in all but one case which had auricular fibrillation associated with fresh myocardial infarction.

**Comment**

Although there are not sufficient cases for statistical evaluation, the data do not suggest that auricular fibrillation is responsible for the mural thrombi in these cases of bacterial endocarditis. The results of the present study indicate the relative rarity of mural thrombi in the heart in cases of bacterial endocarditis with underlying rheumatic heart disease (approximately 10 per cent). This is in contrast to that for the total cases of rheumatic heart disease (27 per cent). These findings may well be a reflection of the demonstrated infrequency in cases of bacterial endocarditis of auricular fibrillation and of severe mitral stenosis. The role of these two factors in the formation of left auricular mural thrombi has been discussed previously.

These findings further substantiate the origin of emboli from the valvular vegetations, rather than from the cardiac chambers despite the bland character of most of the infarcts. The data indicating the source of these emboli from nonbacterial thrombotic vegetations on the valves has been presented previously.

**Summary and Conclusions**

The relative infrequency of mural thrombi in the chambers of the heart is reported in 82 cases of bacterial endocarditis superimposed on rheumatic heart disease and in 47 cases of bacterial endocarditis without underlying rheumatic heart disease.

The relevant literature is reviewed and the possible explanation of this finding is presented on the basis of infrequency of auricular fibrillation and of severe mitral stenosis in cases of bacterial endocarditis (both related to the formation of mural thrombi).

**Sumario Español**

La rareza relativa de trombos murales en las cámaras del corazón se informa en 82 casos de endocarditis bacteriana sobreputa en enfermedad reumática del corazón y en 47 casos de endocarditis bacteriana sin enfermedad cardíaca reumática subyacente.

La literatura pertinente se revisa y la posible explicación de este hallazgo se presenta a base de la rareza de la fibrilación auricular y de la estenosis mitral severa en los casos de endocarditis bacteriana (ambos relacionados a la formación de trombos murales).

**References**

2. Wallach, J. B., Lukash, L., and Angrist, A.


