CLINICOPATHOLOGIC CORRELATIONS

Mitral Insufficiency Secondary to Aortic Valvular Bacterial Endocarditis

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SUMMARY

The anterior leaflet of the mitral valve is partly continuous through a fibrous membrane with the aortic valve. Lying immediately subjacent to the aortic valve, the mitral valve and its chordae may become secondarily infected in instances of primary aortic valvular bacterial endocarditis. Destructive changes of the anterior mitral leaflet or mitral chordae may lead to mitral insufficiency. The latter may dominate the functional and clinical pictures. In some instances, when infection is overcome, the clinical picture may appear to be that of primary mitral valvular disease.

MITRAL INSUFFICIENCY which complicates bacterial endocarditis may occur either in the active or in the bacteriologically healed stage of the disease. Moreover, the mitral infection leading to incompetence of the valve may be an expression of one of three possible settings as follows: (1) isolated infection of the mitral valve, (2) simultaneous infection of the mitral and other valves (usually the aortic), or (3) infection of the mitral valve secondary to primary involvement of the aortic valve.

It is with the latter situation that this communication is concerned.

Mitral insufficiency secondary to primary aortic valvular bacterial endocarditis may result either from mechanical factors concerned with left ventricular enlargement, the latter a result of chronic aortic insufficiency, or from specific destruction of anatomic elements of the mitral valve by infection of these structures as complications of the aortic valvular infection. The tendency for secondary mitral valvular involvement is predicated upon the close anatomic relationships between the aortic and mitral valves. These will first be considered.

Part of the aortic root makes direct connection with the left ventricular wall. The remainder, that related to the adjacent halves of the posterior (noncoronary) and left aortic cusps, makes indirect connection with the left ventricular wall. It does so by the attachment of the aortic root to a fibrous membrane, the aortic-mitral intervalvular fibrosa. The latter membrane, while attaching to the aorta, above, is fused with the base of the anterior leaflet of the mitral valve, below. (Wedged between the aorta and the related left atrial wall is epicardial tissue which extends inferiorly to the aortic-mitral intervalvular fibrosa.) As the intervalvular fibrosa blends with the anterior mitral leaflet and the root of the aorta, the latter is attached indirectly to the left ventricular wall by chordae tendineae and papillary muscles which support the anterior
Bacterial endocarditis of aortic valve with direct extension onto mitral valve and perforation of latter. (a.) Outflow tract of left ventricle (L.V.) and aortic valve. Vegetations of bacterial endocarditis are present in the aortic valve. The subjacent mitral valve shows a perforation (between arrows) resulting from direct extension of infection onto the latter. (b.) Left atrium (L.A.) and a portion of the mitral valve. The perforation (between arrows) seen in a. lies at the junction of the anterior and posterior leaflets. An aneurysm (A.) of the anterior mitral leaflet is also present.

Bacterial endocarditis of aortic valve with nonperforated aneurysm of anterior mitral leaflet. (a.) Left ventricle (L.V.) and bicuspid aortic valve. The cusp adjacent to the anterior mitral leaflet (A.M.) is involved by bacterial endocarditis. Direct extension of infection onto the basal aspect of the anterior mitral leaflet, though perforation through the latter has not occurred. At the lowermost aspect of the ventricular face of the anterior mitral leaflet is a small depression (between arrows) representing the ventricular aspect of an aneurysm of the anteromedial leaflet. (b.) Left atrium (L.A.) and left ventricle (L.V.). A lobulated nonruptured aneurysm (between arrows) involves the lowermost aspect of the anterior mitral leaflet.
Aortic valvular endocarditis with perforation of anterior mitral leaflet. (a.) Aortic valve and outflow tract of left ventricle (L.V.). Numerous perforations in a cusp of aortic valve are the result of bacterial endocarditis. The anterior mitral leaflet shows a perforation (arrow). (b.) Mitral valve opened. The perforation (between arrows) seen in a. is shown involving the anterior leaflet (A.M.). In addition, chordae tendineae show knobby thickenings representing healed vegetations. Hooding of commissural valvular tissue (C.) between anterior and posterior (P.M.) mitral leaflets the result of ruptured chordae. The stump of one of the latter is at point of single arrow.

Figure 3

Mitral leaflet. The anatomic relationship described indicates that the anterior mitral leaflet lies immediately subjacent to the aortic valve and, at lower levels, lie the chordae.

With the foregoing anatomic background, it becomes easily apparent as to ways in which the mitral valve may become infected as a complication of primary infection of the aortic valve. It may do so through one of two mechanisms as follows: (1) direct extension of the infection of the aortic valve inferiorly along the intervalvular fibrosa and onto the anterior mitral leaflet or (2) impingement by blood regurgitating through the aortic valve onto either the ventricular surface of the mitral valve or the chordae tendineae.

Direct extension of infection onto the anterior mitral leaflet may destroy tissue of the latter and lead to a perforation at the basal aspect of the leaflet. Mitral regurgitation may occur through such a perforation (fig. 1).

Complications of impingement of regurgitant blood depend upon the site of impingement. If regurgitating and infected blood strikes the anterior mitral leaflet, the destructive effects of the secondary infection may cause weakness of the involved mitral leaflet. The weakness, in turn, may lead to an aneurysm of the anterior mitral leaflet (fig. 2). Rupture of the aneurysm leads to a perforation of the leaflet and mitral insufficiency (fig. 3).

If the infected blood which regurgitates through the aortic valve is directed more inferiorly, secondary infection with destruction and rupture of chordae tendineae may occur. Usually, the chordae inserting into the anterior leaflet are more prone to involvement...
but in exceptional cases chordae which support the posterior leaflet may become infected and ruptured. The obvious result of ruptured chordae is a flail leaflet and mitral insufficiency (fig. 4).

An uncommon and indirect cause of mitral insufficiency resulting from aortic bacterial endocarditis was described in an earlier communication by the author.1 This was the phenomenon of coronary embolism with resulting gross myocardial infarction and, as a complication of the latter, rupture of a left ventricular papillary muscle.

Certain generalizations may be made regarding the subject of mitral insufficiency resulting from destructive lesions representing complications of bacterial endocarditis of the aortic valve. These are (1) infection of the mitral valve occurs during the active stage of aortic valvular infection; (2) the destructive disease may have a greater effect upon the mitral than upon the aortic valve; and (3) when the foregoing applies and the infection is overcome, patients may present with problems which appear to be those of primary mitral valvular disease.

Reference
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