Valve Ring Abscess in Active Infective Endocarditis

Frequency, Location, and Clues to Clinical Diagnosis from the Study of 95 Necropsy Patients

Ernest N. Arnett, M.D., and William C. Roberts, M.D.

SUMMARY Analysis of 95 necropsy patients with active infective endocarditis (AIE) involving 128 native cardiac valves (aortic = 59, mitral = 48, tricuspid = 20, and pulmonic = 1) disclosed 27 patients with ring abscesses involving 30 valves: the aortic valve ring was infected in 24 patients and only an atrioventricular valve ring (mitral in two, and tricuspid in one) in three patients. Comparison of the following parameters showed no significant differences between the 27 patients with and the 68 patients without ring abscess: age, sex, antibiotic treatment or length of treatment, status of the cardiac valve(s) before infection, and the kind of infecting organism. Comparison of the following parameters, however, showed significant (P < 0.05) differences between the 27 patients with and the 68 patients without valve ring abscess: 1) infection of the aortic valve; 2) occurrence of valvular regurgitation of recent origin; 3) presence of pericarditis; 4) presence of high degree of atrioventricular block; and 5) short duration of symptoms leading to severe debility or death. These five features, therefore, serve as clinical clues to the presence of valve ring abscess in patients with AIE.

IN RECENT YEARS it has become well established that certain patients with active infective endocarditis (AIE) require cardiac valve replacement before completion of adequate antibiotic therapy. The most frequent indication for early valve replacement is congestive heart failure from valvular destruction. A major determinant of outcome in patients undergoing cardiac valve replacement during AIE is the presence or absence of a valve ring abscess. If limited to the valve leaflets or chordae, the infective process usually can be totally excised and the valve prosthesis consequently can be inserted into a sterile field. If valve ring abscess is present, however, the infection may persist behind the site of attachment of the prosthesis, or significant paravalvular leak may follow valve replacement. Although several reports have described the problems of cardical valve replacement in patients with AIE, few have described the frequency of, or location of, valve ring abscess in patients with AIE. The present study analyzes the frequency and location of ring abscesses in a large group of necropsy patients with AIE, and determines whether certain clinical features in these patients might be clues to diagnosis of valve ring abscess during life.

Patients Studied, Methods, and Results

The hearts and clinical and necropsy records of all necropsy patients with AIE accessioned in the Section of Pathology, National Heart and Lung Institute, were re-examined. The anulus of each valve containing a vegetation was sectioned at approximately right angles to search specifically for the presence of a ring abscess. A total of 135 patients were analyzed: 95 had AIE involving natural (unoperated) cardiac valves (observations in 57 of them have been reported elsewhere); 22 had infection at sites of attachment of prosthetic cardiac valves (prosthetic endocarditis) (unpublished observations); six had AIE associated with intracardiac or great vessel shunts; four had AIE after cardiac valve commissurotomy or anuloplasty operations; and eight had AIE involving only mural endocardium. The present analysis concerns only the 95 patients with AIE involving native (unoperated) cardiac valves. Certain clinical and morphologic features in them are summarized in table 1. Of the 95 patients with AIE analyzed, 11 had vegetations limited to the right-sided cardiac valves, ten had vegetations involving both right and left-sided cardiac valves, and 74 had vegetations limited to one or both left-sided cardiac valves. Among the 95 patients, 128 valves were infected: aortic in 59 patients; mitral in 48; tricuspid in 20; and pulmonic in one. Valve ring abscess occurred in 27 (28%) of the 95 patients: 24 patients had one valve ring infected, and three patients had abscesses involving two valve rings. Among the 27 patients with ring abscess, the aortic valve ring was infected in 24 (figs. 1–5): isolated in 21; combined with tricuspid valve ring abscess in two, and with mitral in one. The remaining three patients had ring abscess involving only the mitral (two patients) (fig. 6) or tricuspid valve (one patient).

The parameters analyzed in the 95 patients with AIE are shown in table 1. No significant differences were observed among patients with and without ring abscess regarding age, sex, presence or absence of antibiotic therapy or length of therapy, status of the cardiac valve before it became the site of AIE, i.e., whether or not it was previously functionally normal or abnormal, and type of infecting bacterium. Significant (P < 0.05) differences were observed, however, among patients with and without ring abscess regarding duration of symptoms resulting from AIE, presence of valvular regurgitation resulting from valvular destruction by vegetations, evidence of pericarditis, and high degrees, either second or third degree, of atrioventricular block (table 1). Clinical evidence of AIE was present for less than six weeks in 19 (70%) of the 27 patients with and in 31 (46%) of the 68 patients without ring abscess associated with AIE (P < 0.05). Valvular regurgitation secondary to the AIE was present in 100% of the 27 patients with ring abscess, and in just over half (54%) of the 68 patients without ring abscess associated with AIE (P < 0.05).

Pericarditis was present at necropsy in 14 (52%) of the 27 patients with and in four (6%) of the 68 patients without ring abscess (P < 0.05). Of the 18 patients with pericarditis at necropsy, 14 (78%) had a ring abscess and four (22%) did not
**Table 1. Clinical and Morphologic Observations in 95 Nonoperated Necropsy Patients (Pts) with Active Infective Endocarditis (AIE): Relation to Ring Abscess**

<table>
<thead>
<tr>
<th>Location of Vegetations*</th>
<th>Aortic‡ (59 pts)</th>
<th>Mitral‡ (48 pts)</th>
<th>Tricuspid‡ (20 pts)</th>
<th>Totals‡ (95 pts)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Number patients†</td>
<td>24(41)</td>
<td>35(59)</td>
<td>3(6)</td>
<td>43(94)</td>
</tr>
<tr>
<td>Sex – M:F</td>
<td>20(40): 30(60);</td>
<td>9(3): 90(91);</td>
<td>14(100);</td>
<td>9(18); 81(82);</td>
</tr>
<tr>
<td>&lt; 6 weeks</td>
<td>16(39)</td>
<td>11(41)</td>
<td>3(12)</td>
<td>22(88)</td>
</tr>
<tr>
<td>&gt; 6 weeks</td>
<td>8(25)</td>
<td>2(75)</td>
<td>0</td>
<td>22(100)</td>
</tr>
<tr>
<td>Antibiotics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>3(38)</td>
<td>5(62)</td>
<td>0</td>
<td>3(100)</td>
</tr>
<tr>
<td>&lt; 10 days</td>
<td>8(44)</td>
<td>10(56)</td>
<td>2(10)</td>
<td>19(90)</td>
</tr>
<tr>
<td>&gt; 10 years</td>
<td>13(39)</td>
<td>20(61)</td>
<td>1(4)</td>
<td>22(96)</td>
</tr>
<tr>
<td>Valve leak (from AIE)*</td>
<td>24(57)</td>
<td>18(43)</td>
<td>3(11)</td>
<td>24(89)</td>
</tr>
<tr>
<td>Pericarditis*</td>
<td>14(82)</td>
<td>3(18)</td>
<td>1(17)</td>
<td>5(83)</td>
</tr>
<tr>
<td>Previously normal valve</td>
<td>16(44)</td>
<td>20(56)</td>
<td>2(5)</td>
<td>35(95)</td>
</tr>
<tr>
<td>Heart block (2° or 3°)*</td>
<td>4(80)</td>
<td>1(20)</td>
<td>1(100)</td>
<td>0</td>
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<tr>
<td>Infecting Organism</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Staphylococcus</td>
<td>5(36)</td>
<td>9(64)</td>
<td>2(14)</td>
<td>12(86)</td>
</tr>
<tr>
<td>Pneumococcius</td>
<td>7(70)</td>
<td>3(30)</td>
<td>0</td>
<td>6(100)</td>
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<tr>
<td>Non-alpha streptococci</td>
<td>6(60)</td>
<td>4(40)</td>
<td>0</td>
<td>10(100)</td>
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<tr>
<td>Alpha streptococci</td>
<td>2(20)</td>
<td>8(80)</td>
<td>1(17)</td>
<td>5(83)</td>
</tr>
<tr>
<td>Other bacterium</td>
<td>2(40)</td>
<td>3(60)</td>
<td>0</td>
<td>2(100)</td>
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<tr>
<td>Fungus</td>
<td>1(33)</td>
<td>2(67)</td>
<td>0</td>
<td>3(100)</td>
</tr>
<tr>
<td>Negative blood cultures</td>
<td>0</td>
<td>4(100)</td>
<td>0</td>
<td>5(100)</td>
</tr>
<tr>
<td>Absent blood cultures</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>2(100)</td>
</tr>
</tbody>
</table>

All numbers in parentheses represent percentages. All numbers in brackets represent averages.

*P < 0.05 comparing values for patients with to values for patients without ring abscess.
†Among the 95 patients, 128 cardiac valves contained vegetations; 67 patients had AIE involving only one valve, and 28 patients had AIE involving more than one valve.
‡The figures in these vertical columns refer to numbers of patients with vegetations on that particular valve. Thus, patients with vegetations on more than one valve are listed in more than one column.
§These vertical columns apply only to the total number of patients, i.e., 95, and not to the number of valves containing vegetations, i.e., 128. Therefore, the numbers in these 2 columns are not sums of the columns to their left.

(P < 0.05). A pericardial friction rub had been heard during life in eight of the 18 patients with pericarditis at necropsy. The pericarditis in the 18 patients resulted from extension of a valve ring abscess through the cardiac wall into epicardium in 13 patients; from transmural acute myocardial infarction in two patients; from extension of a myocardial abscess into epicardium in one patient; and from uncertain cause(s) in the remaining two patients.

High degrees of atrioventricular block (complete heart block in five and Mobitz type II block in one patient) occurred in five (19%) of the 27 patients with and in one (1%) of the 68 patients without ring abscess associated with AIE (P < 0.05). Of the six patients with high degrees of heart block, five (83%) had a ring abscess and one did not (P < 0.05). The block in the latter patient resulted from severe narrowing of the lumens of the major extramural

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**Figure 1. Hemorrhagic pericarditis from rupture of an aortic valve ring abscess in a 51-year-old woman (72A-22) with Escherichia coli endocarditis. She presented with acute pericardial tamponade and infective endocarditis was never suspected clinically. a) Longitudinal section showing the ring abscess (A) in the angle between the aortic valve, left atrial (LA) wall and anterior mitral leaflet (AML). RV = right ventricle; LV = left ventricle; PS = pericardial space. b) Close-up of the ring abscess behind the left coronary (LC) cusp. CA = coronary artery; V = vegetation.**

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FIGURE 2. Aortic valve ring abscess in two patients. a) This 29-year-old male heroin addict (A67-108) had had symptoms of congestive cardiac failure for only two weeks before death. He arrived at the hospital one day before death and his blood pressure was 135/0 mm Hg and a loud decrescendo aortic diastolic murmur was present. The causative organism was not identified in life, but large colonies of Gram-positive organisms were present in histologic sections of the aortic valve. Histologic section through aorta (Ao), infected aortic valve, anterior mitral leaflet (AML), and left atrial (LA) wall showing the ring abscess (RA). Hematoxylin and eosin stain × 3. b) Aortic valve infective endocarditis in a 25-year-old woman (A68-46). She presented with fever and palpitations and a "scratchy" precordial sound was heard. Thyrotoxicosis was suspected clinically; infective endocarditis was never suspected. This histologic section shows a ring abscess in the subepicardial adipose tissue of the left atrioventricular sulcus. AV = aortic valve cusp; LV = left ventricle. Hematoxylin and eosin stain × 4.

FIGURE 3. Infective endocarditis in a 40-year-old man (A36-95) with previous mild aortic regurgitation from a congenitally bicuspid aortic valve. Chills, fever and symptoms of congestive cardiac failure began one week after a dental procedure. Examination disclosed dermal petechiae, subungual hemorrhages, Osler's nodes and a loud murmur of aortic regurgitation. Blood cultures were positive for beta hemolytic streptococcus. One week after hospitalization he complained of chest pain and electrocardiogram showed ST-T changes of acute pericarditis. Despite antibiotics, signs of left ventricular failure increased and he died 35 days after the onset of symptoms of infection.

coronary arteries from atherosclerosis. The heart block in the other five patients resulted from destruction of the atrioventricular bundle by extension of the ring abscess into the most cephalad portion of the muscular ventricular septum. One patient had complete right bundle branch block from spread of an aortic valve ring abscess through the membranous ventricular septum and destruction of the right side of the most cephalad portion of the muscular ventricular septum (fig. 6). No patient had complete left bundle branch block.

No significant differences were observed among patients with and without ring abscess when comparing specific organisms. Ring abscess occurred in a third of the patients (21 of 61) with AIE due to a so-called virulent bacterium (staphylococcus, pneumococcus, non-alpha streptococcus) and also in a third of the patients (3 of 10) with AIE due to a so-called non-virulent bacterium (alpha streptococcus). Among our 95 patients, however, AIE resulted from alpha streptococcus in only ten patients, and therefore, the AIE in nearly all of the remaining 85 patients probably resulted from infection by so-called virulent organisms.
Figure 4. The heart in a 54-year-old man (A75-55) with infective endocarditis involving a calcified, stenotic, congenitally bicuspid aortic valve. Fever and syncope occurred several weeks after tooth extraction and blood cultures yielded alpha streptococcus. Despite antibiotic therapy, signs of left ventricular failure increased, and he died 23 days after the onset of symptoms of infection. a) Postmortem roentgenogram of the heart showing a large amount of calcium in the aortic valve. RV = right ventricle; LV = left ventricle. b) Longitudinal section through the aortic valve showing a large ring abscess. The abscess has burrowed to the epicardial surface and is contained only by a thin layer of fibrous tissue. Multiple small vegetations are present on the mitral valve chordae, but no chordae are ruptured. The distal third of the anterolateral papillary muscle is necrotic. RA = right atrium; LAA = left atrial appendage. c) Aortic valve viewed from above. The orifice of the valve is markedly stenotic. The ring abscess lies beneath the left main (LM) coronary artery (CA). PT = pulmonary trunk. d) Exterior of the heart showing focal fibrous pericarditis. The pericarditis resulted from extension of the ring abscess into the pericardial space. Ao = aorta.

Figure 5. Staphylococcus epidermis aortic valve endocarditis in a 17-year-old boy (A69-4). Massive aortic regurgitation and right bundle branch block developed during the infection and he died 28 days after the onset of symptoms of infection. a) Opened aortic valve and left ventricle. The right (R) coronary cusp is destroyed by the infection and a ring abscess is present behind this cusp. N and L = non and left coronary cusps; A = anterior mitral leaflet; LV = left ventricle. b) Opened right atrium (RA), tricuspid valve and right ventricle (RV) showing a bulging membranous ventricular septum (dashed circle) with a perforation in its center. c) Longitudinal section through the aneurysm in the ventricular septum (VS). d) Longitudinal section anterior to showing a ring abscess in the muscular portion of the ventricular septum.
**Comments**

The present study of 95 necropsy patients with active infective endocarditis (AIE) resulted in the following firm observations: 1) that ring abscess is common at necropsy in patients in AIE, occurring in nearly one-third of them; 2) that ring abscess is common among necropsy patients with AIE involving the aortic valve but is quite uncommon in patients with AIE isolated to any of the other three cardiac valves; 3) that there are five clinical clues which suggest the presence of a valve ring abscess during life. These include a) AIE involving the aortic valve; b) presence of valvular regurgitation of recent origin; c) evidence of pericarditis; d) evidence of high degrees of atrioventricular block, and e) a short duration of symptoms caused by AIE resulting in severe debility.

Although it occurred in nearly a third of our 95 necropsy patients, ring abscess was common only among the patients with AIE involving the aortic valve. Of 59 patients with vegetations on the aortic valve, ring abscess occurred in 24 (41%), whereas among 36 patients with AIE without aortic valve vegetations, ring abscess occurred in only three (6%). Of the six patients with ring abscess involving either the mitral or tricuspid valves, three also had aortic valve ring abscess. One of the three patients with isolated mitral ring abscess had the infection superimposed on a heavily calcified mitral anulus. Thus, of the clues suggesting the presence of a ring abscess, infection of the aortic valve, rather than any of the other three valves, is nearly a prerequisite.

Valvular regurgitation is always present in patients with valve ring abscess. All of our 27 patients with ring abscess had signs of valvular regurgitation resulting from AIE, i.e., of recent onset, whereas signs of valvular regurgitation were present in only 54% of the 68 patients with AIE without ring abscess. Classical signs of aortic regurgitation, however, may be absent in patients with acute aortic regurgitation, and this observation should be borne in mind regarding any patient suspected of having AIE.

The most common cause of pericarditis in patients with AIE is extension of the valvular ring abscess into epicardium. Thus, the occurrence of a pericardial friction rub or pericardial effusion during the course of AIE is highly suggestive of ring abscess. As early as 1856, Rokitansky recognized that ring abscess may be associated with pericarditis and myocarditis, and that it may cause a cardiac aneurysm which may rupture into a cardiac chamber or pericardial space. Ponick in 1873 again noted the association of AIE with pericarditis, and Osler in 1885 noted the tendency of valve ring abscess to burrow into adjacent structures. Of 43 consecutive necropsy patients with AIE studied by Beck, eight (16%) had pericarditis and five of them had an aortic valve ring abscess with perforation into the pericardial space. McColl reported a patient with pericarditis due to mycotic aneurysm resulting from AIE, and concluded from a review of previously reported cases that perforation of a ring abscess was the most frequent cause of pericarditis in patients with AIE. A similar observation was made by Buchbinder and Roberts. Among 38 patients with aortic valve AIE reported by Utley and Mills, 15 had ring abscess and five of them had pericarditis. Evidence of pericardial disease was evident at necropsy in 18 of our 95 necropsy patients with AIE and 14 of them had a ring abscess. In 13 of the 14 patients the pericarditis was the result of extension of the inflammatory process from the ring abscess into epicardium. Although only half of our patients with evidence of pericardial disease at necropsy were noted to have a pericardial friction rub during life, this sign clearly was not sought in most of the other patients.

Either complete heart block or a high degree of second degree heart block appearing suddenly in a patient with AIE is highly suggestive of the presence of a ring abscess, which
has extended into the area of the atrioventricular node or bundle.\(^{27-31}\) Of our six patients with either complete heart block (five patients) or Mobitz type II atrioventricular block, five had a valve ring abscess. In addition, the appearance of complete right or left bundle branch block during AIE is also highly suggestive of the presence of a ring abscess with extension into the most cephalad portion of the muscular ventricular septum. Although only one of our patients developed complete right bundle branch block and none developed complete left bundle branch block, the latter has been reported in AIE by Roberts and Somerville.\(^{27}\)

In this study, the duration of symptoms attributable to AIE was significantly shorter among patients with ring abscess than among patients without ring abscess. Symptoms of AIE were present for less than six weeks in 70% of the 27 patients with and in 46% of the 68 patients without ring abscess. Utley and Mills\(^{28}\) also found a significantly shorter duration of symptoms leading to death or severe debility among patients with ring abscess, than among patients without ring abscess. The explanation for this shorter symptomatic period is uncertain. The difference could not be attributed to the type of infecting bacterium, or to the presence of a previously normal or abnormal valve. Furthermore, this shorter duration of symptomatic illness could not be attributed to the presence or absence of, or length of, antibiotic administration: 43 patients received either no antibiotics or antibiotics for less than ten days; and 12 (28%) of them had ring abscess; 52 patients received antibiotics for longer than ten days and 15 (29%) of them had a ring abscess.

A study of ring abscess during AIE by Sheldon and Golden in 1951\(^{29}\) emphasized that this complication resulted when the infecting process was due to a highly virulent organism, like pneumococcus and staphylococcus. The infecting organism in each of their 12 patients with ring abscess was caused by one or the other of these two organisms, whereas among their 11 patients with alpha streptococcus endocarditis, none had a ring abscess. Likewise, among 38 patients with aortic valve endocarditis reported by Utley and Mills,\(^{28}\) anular involvement was less common when the infection was due to alpha streptococcus than when it was due to Staphylococcus aureus or non-alpha streptococci. Among our 95 necropsy patients with AIE, the infecting organism, when known, also was usually a highly virulent one. When, however, the patients were grouped according to the presence or absence of ring abscess, no significant differences between types of causative organisms were observed.

References

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Valve ring abscess in active infective endocarditis. Frequency, location, and clues to clinical diagnosis from the study of 95 necropsy patients.

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_Circulation_. 1976;54:140-145
doi: 10.1161/01.CIR.54.1.140

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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