Non-atherosclerotic cardiac manifestations of rheumatoid arthritis

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Pericardial diseases

First clearly described by Charcot in the 19th century, pericardial disease has been regarded as the most common cardiac manifestation of rheumatoid arthritis (RA). Pericarditis has been shown to affect about one-third of RA patients, with some variations in prevalence from 30 to 50% depending on the diagnostic method (i.e., echocardiography versus post-mortem examination) and the calendar period under study [1–4]. While in most cases pericarditis develops after the onset of RA, some studies have demonstrated that it can precede RA diagnosis and may be the first manifestation of RA disease activity. A recent systematic review and meta-analysis of case-control studies from 1975 through 2010 reported that the risk of developing pericardial effusion was tenfold higher in patients with RA compared with non-RA subjects (odds ratio [OR] 10.7; 95% confidence interval [CI] 5.0–23.0) [2].

Pericardial disease in RA is frequently asymptomatic. Only a minority of patients (<10%), most commonly males, with severe rheumatoid factor (RF)-positive and often with nodular RA, develop clinical pericarditis [1,5]. The annual incidence of clinically manifest pericarditis in RA has
been found to be 0.34% in females and 0.44% in males [5]. The majority of symptomatic patients present with acute pericarditis [3]. Progression from exudative to constrictive pericarditis has been described in up to one-fourth of patients [6]. Similar to rheumatoid pleural effusions, pericardial effusions in patients with RA are commonly sterile, with variable leukocyte counts, high protein levels, decreased complement and glucose levels, and presence of RF and immune complexes [2,4,5]. Although rare, cholesterol pericarditis presenting as pericardial effusion with high cholesterol content, and in some cases with progression to constrictive pericarditis, has been described in patients with RA [7]. Symptomatic pericarditis is associated with increased mortality in patients with RA, predominantly in the first year of follow-up [3,8]. The highest mortality rates were observed in RA patients with constrictive and rapidly progressive pericarditis [8]. Chronic pericardial disease has been described in patients with severe long-standing RA, some of them initially presenting with cardiac tamponade requiring urgent surgical management [2,3].

More recently, there has been emerging evidence of the association between treatment with biologic disease-modifying antirheumatic drugs (bDMARDs) and development of pericarditis. While biologic response modifiers (BRM) generally appear to be associated with better control of articular and some extra-articular manifestations of RA including vasculitis, several reports have described development of acute and/or recurrent pericarditis, occasionally with tamponade, in patients treated with tumor necrosis factor (TNF) inhibitors, requiring discontinuation of anti-TNF treatment and often surgical management (ie, pericardectomy, pericardial window, or drain) [9]. Most of these cases have been associated with a non-infective pericardial effusion, which was thought to be due to a paradoxical RA flare in the setting of anti-TNF therapy [9]. However, cases of infective pericarditis with purulent pericardial effusion in RA patients on anti-TNF inhibitors or the B-lymphocyte inhibitor rituximab, with or without combination therapy with traditional disease modifying anti rheumatic drugs (DMARDs), have also been described [10,11]. These patients required prolonged systemic antibiotic therapy.
Diagnosis
Pericardial disease is typically diagnosed with echocardiography. Transthoracic echocardiography (TTE) is performed when there are symptoms or signs suggestive of pericarditis, pericardial effusion, or constrictive pericarditis. A small pericardial effusion is the most common finding. TTE is frequently used as the initial diagnostic tool used to establish the diagnosis of constrictive pericarditis; however, alternative imaging may be required to help aid in the diagnosis. Cardiac magnetic resonance (CMR) imaging is able to diagnose pericardial effusions, pericardial thickening (Figure 2.1), and pericarditis, using special techniques that identify inflammation (Figure 2.2A and B).

Treatment
Asymptomatic pericardial effusions can be self-limiting, particularly when the effusion is small. Mild symptomatic pericarditis usually responds well to treatment with aspirin or non-steroidal anti-inflammatory drugs (NSAIDs), primarily ibuprofen [12]. Glucocorticoid therapy may be used

![Figure 2.1 Magnetic resonance imaging in a patient with rheumatoid arthritis. Axial double inversion recovery sequence demonstrates pericardial thickening, measuring 6 mm (arrow). Courtesy of Dr Crystal Bonnichsen, Division of Cardiovascular Diseases, Mayo Clinic, Rochester, Minnesota.](image)
Figure 2.2 (A) Short axis delayed enhancement image shows bright enhancement of the pericardium (arrows) consistent with pericardial inflammation.

Figure 2.2 (B) Triple inversion recovery short axis image in the same patient shows increased signal in the pericardium (arrow), which indicates pericardial edema and active inflammation. Courtesy of Dr Philip Araoz, Department of Radiology, Mayo Clinic, Rochester, Minnesota.
in more severe disease, in cases of contraindications to aspirin/NSAIDs, or failure of one or more NSAIDs. The use of low-to-moderate doses of glucocorticoids is recommended (ie, prednisone 0.2–0.5 mg/kg/day). Treatment with either NSAIDs or glucocorticoids should continue until complete resolution of symptoms and normalization of inflammatory marker levels. Glucocorticoids should be tapered off slowly, after remission has been achieved. Colchicine can be used in addition to aspirin, NSAIDs, or glucocorticoids to improve disease control and decrease the rate of recurrence. There is weaker evidence to support the use of other treatment regimens especially in patients with recurrent or refractory disease, including azathioprine and other immunosuppressive drugs, intravenous immunoglobulin, or other bDMARDS such as interleukin 1 (IL-1) inhibitors for example, anakinra [13]. The use of these agents should be limited to cases where conventional medications have failed to produce adequate disease control [12].

While most cases of RA-related pericarditis can be effectively managed with medications, some severe cases associated with cardiac tamponade, and hemodynamically significant pericardial effusion, constrictive or effusive-constrictive physiology, and chronic or treatment-refractory pericarditis may require surgical management. Invasive techniques include pericardiocentesis, pericardiectomy, or pericardiotomy (‘window’ pericardiectomy) [12].

**Myocardial diseases**

Compared with pericardial disease, myocardial involvement in RA is less common. Post-mortem studies report findings consistent with either diffuse or focal myocarditis in 11–20% patients with RA [14,15]. However, most RA patients with myocarditis are clinically asymptomatic and the disease follows a self-limiting course. Symptomatic myocarditis is rare and is usually associated with active articular disease and other extra articular disease manifestations of RA (ExRA). In these more severe, clinically manifest cases, complications such as secondary cardiomyopathy and resulting heart failure (HF) as well as cardiac arrhythmias and conduction abnormalities can occur [16,17].
The prevalence of RA-associated cardiomyopathy is not well understood and large cohort studies on the subject are lacking. A small case series of 30 patients with RA suggested a 37% prevalence of cardiomyopathy based on echocardiography studies [18]. Both ischemic and secondary non-ischemic cardiomyopathies resulting from either non-specific or granulomatous myocarditis or anti-rheumatic medication use, such as glucocorticoids and antimalarial medication, have been described in RA [4].

Myocarditis in RA follows one of the two histological patterns:
1. a granulomatous form that is considered specific for RA, with morphological features typical for subcutaneous RA nodules and predilection for the myocardium of the left ventricle (LV); and
2. a non-specific form that is also observed in other disorders and is morphologically characterized by interstitial infiltration with lymphocytes, plasma cells, and histiocytes [14].

**Imaging and myocardial disease**

Among imaging studies, echocardiography has been commonly used to assess the LV ejection fraction (EF) in patients with concern for myocardial involvement; however this technique is not able to detect myocardial inflammation and thus appears to be unsuitable for detection of myocarditis. CMR imaging has been evolving as a useful non-invasive modality for early diagnosis of myocardial tissue abnormalities providing results similar to myocardial biopsy [19].

A few characteristic imaging features of myocardial involvement have been suggested in patients without cardiovascular symptoms but with active RA based on standardized CMR study. These include an increased T2-weighted edema ratio (ER) score suggesting myocardial tissue edema, myocardial wall thinning, and cavity dilatation, with reduced global EF and globally raised late gadolinium enhancement (LGE) scores, potentially reflecting diffuse myocardial fibrosis resulting from microvascular disease, macrovascular CAD, and chronic myocardial inflammation [20]. With the introduction of higher resolution techniques for improved myocardial imaging, such as 3- and 7-Tesla scanners, the diagnostic possibilities for myocardial imaging may expand significantly in the near
future, with corresponding opportunities for earlier recognition and timely management of myocardial involvement in RA [19].

**Treatment**
Due to the rare occurrence of rheumatoid myocarditis, optimal treatment has not been established and evidence-based guidelines on management of myocarditis in RA are lacking. Conventional cardiovascular treatment aimed at support of LV function in conjunction with aggressive anti-inflammatory therapy and treatment with DMARDs theoretically should be beneficial. High-dose glucocorticoid therapy has been associated with resolution of imaging abnormalities, normalization of LV function and resolution of HF symptoms in a patient with rheumatoid vasculitis and myocardial involvement [21]. The resolution of conduction abnormalities associated with myocarditis in RA may also occur when the underlying RA disease is well-controlled [22].

Azathioprine and cyclophosphamide can be used in RA patients who did not respond to high-dose glucocorticoids. The role of bDMARDs (particularly anti-TNF agents) in the management of rheumatoid myocarditis remains uncertain, which is in part due to concerns for potential harm associated with anti-TNF agents use in advanced HF [23].

**Antimalarial agents and cardiomyopathy**
Over the past two decades there has been growing evidence of the association between the use of antimalarial agents, primarily chloroquine and hydroxychloroquine, and myocardial toxicity, clinically manifesting as HF in the setting of restrictive or dilated cardiomyopathy and/or conduction abnormalities in RA patients [24]. The exact mechanism of the cardiomyopathy associated with the use of antimalarials remains unclear but may involve lysosomal changes and accumulation of glycogen and phospholipids resulting in a vacuolar type of myopathy [24] including enlarged and vacuolated cells on light microscopy. Electron microscopy processing shows cytoplasmic granules with curvilinear and myelin-like configurations (‘myeloid bodies’), thought to be abnormal lysosomes, pathognomonic to antimalarial cardiotoxicity [24,25]. In the absence of specific treatment, prognosis in antimalarial cardiotoxicity ranges from
partial or complete recovery of cardiac function with early recognition of antimalarial toxicity and timely withdrawal of the offending drug to heart transplantation or death in unrecognized cases.

**Cardiac amyloidosis**

Cardiac amyloidosis is a rare manifestation of systemic RA-associated amyloid A (AA)-amyloidosis. Exact estimates of incidence and prevalence of cardiac amyloidosis are lacking, which is in part due to unrecognized disease. Myocardial infiltration with amyloid and progressive myocardial fibrotic changes results in myocardial hypertrophy, impairment of systolic and diastolic function in the setting of restrictive cardiomyopathy, and subsequent unfavorable cardiovascular outcomes and increased mortality [19].

While histological diagnosis is a gold standard for the diagnosis of cardiac amyloidosis, its use is limited due to the invasive nature of myocardial biopsy. Increased echogenicity of the myocardium with a granular or ‘sparkling’ appearance on TTE has been associated with a diagnosis of cardiac amyloidosis. However, these myocardial characteristics have been found in other causes of LV hypertrophy, and despite high specificity (up to 80%) the sensitivity of this pattern of echocardiographic findings for diagnosis of cardiac amyloid has been generally low (about 30%) [26,27]. Several studies have demonstrated a pattern of LGE on CMR imaging, suggestive of cardiac amyloid depositions, in patients with systemic amyloidosis [28]. Thus, newer modalities of CMR may be promising in means of identifying early phenotypical features of cardiac amyloidosis.

The prognosis of patients with cardiac amyloidosis is generally poor due to associated progressive HF. The use of cytotoxic drugs and bDMARDs can help to temporarily improve cardiac function [29,30]. However, progressive organ failure can occur despite aggressive treatment.

**Non-atherosclerotic coronary artery disease**

In addition to atherosclerotic disease, myocardial ischemia can occur in RA patients secondary to vasculitis. Coronary vasculitis is often associated with the rheumatologic vasculitides but can also occur in RA [31]. The term rheumatoid vasculitis (RV) has been coined to refer to the
development of inflammation in the wall of the blood vessels in patients with RA. Typically, this process involves the medium-sized muscular arteries through to the smaller arterioles or post-capillary venules. Rheumatoid vasculitis tends to occur in those patients with longer standing, erosive disease and manifests in those patients who have had RA for more than 10 years. An early autopsy study places the incidence of epicardial vasculitis at 20%. In this study, the affected vessels identified as involving vasculitis were in various stages of healing, and of variable clinical significance [31,32]. Vasculitis of the epicardial arteries can lead to ischemia similar to epicardial atherosclerotic disease. Moreover, vasculitis of the small intramural cardiac vessels can lead to myocardial ischemia, and vasculitis of the vaso-vasorum can result in epicardial vessel dysfunction [32]. Vasculitis can also promote the development of atherosclerotic disease in affected vessels [33]. Overall, rheumatoid vasculitis has ranging levels of severity and clinical manifestations but can be associated with greater morbidity and mortality than the RA itself.

**Arrhythmias**

Arrhythmias are a significant component of the cardiac manifestations of RA and are an area of ongoing research. There is an increased risk of sudden cardiac death in patients with RA compared with the non-RA cohort [34]. Arrhythmias in RA patients may be due to the RA process itself or, more commonly, due to ischemic disease, heart failure, or amyloid deposition.

**Atrial arrhythmias**

The role of RA in the development of atrial fibrillation (AF) is an area of debate. Although population-based studies have conflicting attributable risk of AF to RA, there are several risk factors for AF that are more common in the RA population. In the general population there is an increased risk of the development, recurrence, and persistence of atrial fibrillation in patients with underlying inflammation. This has been specifically demonstrated in patients with elevated TNF-alpha, IL-2, IL-6, and C-reactive protein (CRP) [35,36]. Based on these findings it would seem likely that patients with RA, particularly untreated or undertreated
RA, would have an increased risk of developing AF. Patients with RA have increased p-wave dispersion (PWD) [37]. PWD is a measure of the difference of p wave duration on a surface electrocardiogram and represents inhomogeneous conduction through the atria. In the non-RA cohort, increased PWD is a predictor of AF [38]. In the RA population, PWD correlates with CRP levels, suggesting increased inflammation may increase a patient’s risk of AF [37]. However, in the RA population PWD has not been demonstrated to correlate with the prevalence of AF. In the general population, there is evidence that increased inflammatory markers are associated with an increased risk of AF, and that controlling inflammation can reduce the incidence [34]. Again, this has not been demonstrated in the RA population.

There have been two major studies examining the prevalence of AF in patients with RA. A Danish study found an overall incidence of AF that was 40% higher in the RA cohort than in the general population [39]. A health claim database study and another population based study from the United States found no increase in incidence of AF in patients with RA after adjusting for potential confounders [40, 41].

**Conduction system disease**

In a review of electrocardiograms (ECGs) of patients with RA, the RA group had slightly longer PR intervals than their non-RA peers but the mean PR intervals in both groups were within normal limits [42]. In the same study there was a low prevalence of low degree atrioventricular (AV) blocks in the RA cohort. Most of the AV blocks in RA patients were third degree blocks [22]. Complete AV nodal blocks in RA patients have been associated with AV nodal infiltration by rheumatoid nodules, mononuclear cells, extension of inflammatory lesions from valvular involvement, and amyloid deposition [4, 22]. Medications for RA have also been implicated in the development of complete heart block, including chloroquine.

Beyond the atrioventricular node, rheumatoid nodules can contribute to additional conduction system disease [4]. However, treating the underlying RA disease process with immunosuppressive therapies has not been shown to treat the arrhythmias due to rheumatoid nodules. In addition, pro-inflammatory cytokines, particularly TNF-alpha, may
prolong myocyte action potential, predisposing to re-entrant ventricular arrhythmias [34].

Fibrosis secondary to RA can involve the conduction system, resulting in arrhythmias. Additionally, antibodies to cardiac conducting tissue, including Purkinje cells, can contribute to arrhythmia development [22].

**Ventricular arrhythmias**

Patients with RA are twice as likely to experience sudden cardiac death (SCD) as those without RA, even after adjusting for a history of myocardial infarction [43]. The mechanism of this increased incidence of SCD in RA patients remains unclear, but there is evidence that it may be due to the role of systemic inflammation in RA beyond atherosclerotic disease as well as autonomic dysfunction in RA patients [34].

Systemic inflammation, particularly inflammation within ventricular tissues, may lead to a delay in ventricular repolarization. This is manifest as an increased QT interval on an ECG, which is indeed seen more commonly in RA patients. One study found that after the diagnosis of RA, patients were more likely to develop prolonged heart rate corrected QT interval (QTc) than patients who were not diagnosed with RA [44]. Similarly, elevated CRP and positive anti-CCP are associated with prolonged QTc. Erythrocyte sedimentation rate (ESR) and RF have also been shown to trend positively toward prolonged QTc [45]. Pro-inflammatory cytokines, particularly TNF-alpha, may prolong myocyte action potential, predisposing to re-entrant ventricular arrhythmias, although this has not been specifically demonstrated [34]. Interestingly, patients with RA and prolonged QTc have increased all-cause mortality relative to their RA peers without prolonged QTc, but no difference in cardiovascular mortality compared to their RA peers [44,44]. This discrepancy suggests there may be another mechanism leading to increased ventricular arrhythmias and sudden cardiac death in the RA population.

Additional research into the mechanism for increased sudden cardiac death in patients with RA focuses on increased sympathetic tone in RA patients [34]. Heart rate variability is often used as a marker of sympathetic drive and parasympathetic suppression, with decreased heart rate variability indicating increased sympathetic drive. RA patients demonstrate
decreased heart rate variability compared with non-RA peers [46]. In one study, women with RA were found to have a higher resting heart rate compared with their non-RA peers, suggesting decreased vagal tone as well as decreased heart rate variability, further pointing to a higher sympathetic drive. The increased sympathetic drive may be due to the so-called ‘inflammatory reflex’ in which pro-inflammatory cytokines target the autonomic centers of the brain. This, in turn, increases sympathetic drive to ultimately inhibit cytokine production and self-regulate inflammation [46]. There are currently no anti-arrhythmic therapies that specifically address arrhythmias in the RA population. Treatment of arrhythmia in a patient with RA should focus on the underlying rhythm disturbance [22].

Valvular heart disease

Valvular heart disease (VHD) is a known extra-articular complication of RA [2]. However, there is much uncertainty regarding the prevalence and pathophysiology of VHD in RA. There are several challenges to studying valve disease in RA patients. The majority of VHD in RA patients is asymptomatic, making prevalence studies difficult to conduct [2,18]. The prevalence of VHD in RA is significantly higher in autopsy studies than in echocardiographic studies. Additionally, as cardiac imaging technology advances, the ability to characterize subclinical valve disease will increase. VHD in RA presents in multiple forms, and has an inconsistent relationship with a systemic inflammatory disease course, making it difficult to select patients for studies.

Mechanisms of valvular heart disease

There are three major categories of valve destruction in RA: fibrosis, calcification, and granuloma formation. All three pathologies can be found on any of the cardiac valves, however, RA VHD is significantly more common on the left-sided valves, and most studies suggest that the mitral valve is most commonly affected.
Valvular fibrosis

Valve thickening is the most commonly described valvular pathology in RA. Autopsy and surgical pathology studies find thickening and fibrosis of each of the cardiac valves in RA patients, although the mitral valve is most commonly involved (Figure 2.3 and 2.4). Acute or recurrent valvulitis can cause thickening of the valve and is the result of infiltration of the valve tissue with plasma cells, histiocytes, lymphocytes, and eosinophils producing valve fibrosis, and, ultimately, valve retraction [47]. Echocardiographic studies have demonstrated a similar frequency of focal and diffuse involvement of valvulitis in patients with RA. Focal valvulitis affects the base, mid, and tip portions of the valves equally, and less commonly affects the mitral annulus or chordae tendineae. Valvular fibrosis in RA patients is similar to natural valve aging described in the general population, and, indeed, some patients with RA may demonstrate both RA-driven valvulitis in addition to aging or atherosclerotic valve fibrosis. No relationship has been identified between the presentation of valve fibrosis and the duration of inflammatory disease. Rheumatoid aortitis (extremely rare in the present day) can occur and may lead to

Figure 2.3 Pathologic specimen of an aortic valve in a patient with rheumatoid arthritis. Valve thickening (single arrow), and commissural fusion (double arrow). Courtesy of Dr William Edwards, Department of Laboratory Medicine and Pathology, Mayo Clinic, Rochester, Minnesota.
aortic valve regurgitation (along with aortic aneurysms). Typically, this has been reported post-mortem [48]. Treatment with high-dose glucocorticoids is recommended initially [48].

**Valvular calcification**

Similar to valvulitis, calcification of the valves can result in both regurgitation and stenosis. Calcification can be present on any of the cardiac valves, but in the RA population is more commonly found on left-sided valves. In the general population, both cardiac valvular and arterial calcification are thought to be related to inflammation, although the exact mechanism remains unclear. A study using multi-detector computed tomography noted that patients with RA have a higher prevalence of both mitral and aortic valve calcification relative to their non-RA peers. Similar to the non-RA cohort, the presence of aortic and mitral valve calcification increases with age, although valve calcification is seen at younger ages in the RA population compared with the non-RA population. Age and duration of disease have been demonstrated as independent predictors
of mitral valve calcification in RA patients. CRP has not been shown to be related to the presence of valvular calcification [49].

**Rheumatoid nodules**

Unlike fibrosis and calcification, rheumatoid nodules are more specific to systemic inflammatory diseases, particularly RA. Rheumatoid nodules in the heart are significantly less common than articular rheumatoid nodules but have been described on all four cardiac valves (Figure 2.5A and B). Rheumatoid nodules are most commonly found within and at the base of leaflets and valve rings, and less commonly on papillary muscles or endocardium. The presence of rheumatoid nodules on cardiac valves results in both stenosis and regurgitation (Figure 2.6). There is limited evidence that some cardiac rheumatoid nodules may improve with steroid therapy [50].

**Non-infectious endocarditis**

Although often associated with other rheumatologic conditions, non-infectious or autoimmune endocarditis is a particularly devastating complication of RA. It is a significant cause of valve damage and embolic phenomena. Autoimmune endocarditis is characterized by antibody-initiated damage and activation of the endothelium, followed by inflammatory cell infiltration with T cells and macrophages. The condition nonbacterial thrombotic endocarditis (NBTE) in which vegetations composed of platelet-fibrin thrombi develop on the valve, has also been described in RA. A specific subset of NBTE is Libman-Sacks endocarditis, which is commonly associated with systemic lupus erythematosus and antiphospholipid syndrome, but can also be present in RA. NBTE can result in valvular dysfunction or embolic disease requiring valve repair or replacement, although it can also be asymptomatic and identified only at autopsy [51].

**Relationship to disease activity**

Patients may have cardiac involvement with their RA but without cardiac symptoms or evidence of other extra-articular manifestations of RA. One of the challenges in characterizing VHD in RA patients is that disease
Figure 2.5 (A) Transthoracic echocardiogram, parasternal long axis view. Mitral valve nodule (arrow) on the tip of the anterior mitral valve leaflet in a patient with rheumatoid arthritis. (B) Apical long axis view in the same patient, zoomed up on the mitral valve. Anterior mitral valve leaflet with evidence of a nodule (arrow).
activity is variable over time, but valvular disease is progressive. Thus, cross-sectional studies designed to evaluate the relationship between disease severity and the presence of VHD are forced to evaluate disease activity based on current markers of disease activity as opposed to lifetime cumulative disease activity. Several studies have failed to consistently identify any markers of disease activity or demographic characteristics that correspond to VHD [52].

**Valve surgery in rheumatoid arthritis**

Although much of the valvular heart disease in RA patients is asymptomatic, some patients will be hemodynamically compromised secondary to their valve disease and require surgery. Surgical treatment of VHD in RA patients requires a team approach, with involvement of the patient’s rheumatologist, cardiologist, and cardio-thoracic surgeon. When a valve requires surgical replacement the health care team must select between a bioprosthetic or a mechanical valve. Mechanical valves provide a durable, long-lasting prosthesis. However, they promote valvular
thrombotic vegetations and therefore require lifelong anticoagulation. This can be particularly problematic in patients with RA as these patients may require several joint surgeries, and they are prone to secondary antiphospholipid syndrome, with the increased risk for both thrombotic and bleeding complications. Alternatively, bioprosthetic valves have the benefit of not requiring long-term anticoagulation, which is favorable in patients who will need future orthopedic surgeries secondary to erosive RA. However, bioprosthetic valves have a limited life span, which may be even shorter in RA patients due to systemic inflammation. Data on cardiac surgery in antiphospholipid syndrome are limited to small case series, with conflicting results on whether it is better to replace with a bioprosthetic or a mechanical heart valve [53]. Many patients with RA have known antiphospholipid antibodies without overt antiphospholipid syndrome. The role of the presence of these antibodies in selecting a cardiac valve is unclear.

In addition to selecting a cardiac valve, perioperative management should include a multidisciplinary approach to managing both RA and cardiac symptoms. A database study that reviewed mitral valve procedure outcomes in RA patients found that RA patients have a similar length of stay and mortality relative to their non-RA peers undergoing similar procedures [54]. The presence of RA is not a contraindication to undergoing valve surgery.

References


Handbook of Cardiovascular Disease Management in Rheumatoid Arthritis
Semb, A.G. (Ed.)
2017, XVI, 109 p. 28 illus., 16 illus. in color., Softcover
ISBN: 978-3-319-26780-7