CARDIOLOGY Rounds®

As presented in the rounds of the Division of Cardiology, St. Michael's Hospital, University of Toronto

An Overview Of Cardiac Sarcoidosis

By FAHAD BASLAIB MD, FRCPC and GORDON MOE MD, FRCPC

Sarcoidosis, a multisystem disorder affecting individuals worldwide, is pathologically characterized by the presence of noncaseating granulomas in involved organs. Although environmental and genetic factors have been implicated in its pathogenesis, the etiology of cardiac sarcoidosis remains obscure. The disease typically affects young adults and presents with one or more of the following abnormalities: bilateral hilar adenopathy; pulmonary reticular opacities; and skin, joint, and/or eye lesions. Cardiac involvement in patients with sarcoidosis is increasingly recognized and associated with poor outcomes. Although clinical evidence of myocardial involvement is present in about 5% of patients with sarcoidosis, autopsy studies indicate that subclinical cardiac involvement occurs in 20% to 30% of cases.[1,2] This issue of *Cardiology Rounds* reviews the epidemiology, pathology, clinical diagnosis, and treatment of cardiac sarcoidosis.

Epidemiology

Sarcoidosis is more commonly seen in young and middle-aged adults, and more females are affected than males. In the United States (US), the prevalence of sarcoidosis ranges from 10.9 per 100,000 in the white population to 35.5 per 100,000 in the black population. In Europe, Scandinavians have one of the highest incidence rates with 50 to 60 cases per 100,000 population.[3] Myocardial involvement occurs in at least 25% of patients with sarcoidosis in the US, and accounts for 13% - 25% of deaths from sarcoidosis.[4] In Japan, sarcoid heart disease is more common and is responsible for up to 85% of the deaths from sarcoidosis.[5]

Pathologic features

No part of the heart is immune to infiltration by sarcoid granulomas. Granulomas may involve the pericardium, myocardium, and endocardium, but of the three, the myocardium is the part most frequently involved. In decreasing order of frequency, the predominant sites of myocardial involvement are the left ventricular free wall and papillary muscles, the basal aspect of the ventricular septum, the right ventricular free wall, and the atrial walls.[6] Samples of myocardium with sarcoidosis involvement reveal the presence of numerous lymphocytes located in the border zones around the granulomas. A dense band of fibroblasts, collagen fibres, and proteoglycans usually encase this aggregate of inflammatory cells (Figure 1).[7]

Clinical manifestations

Cardiac manifestations of sarcoidosis may precede, follow, or occur at the same time as other systemic symptoms of the disease, such as:

- conduction abnormalities
- ventricular arrhythmias
- heart failure
- supraventricular arrhythmias
- valvular dysfunction
- simulated myocardial infarction.

Division of Cardiology

Beth L. Abramson, MD
Abdul Al-Hesayen, MD
Luigi Casella, MD
Thierry Charron, MD
Asim Cheema, MD
Robert J. Chisholm, MD
Chi-Ming Chow, MD
Paul Dorian, MD
David H. Fitchett, MD (Assoc. Editor)
Michael R. Freeman, MD
Shaun Goodman, MD
Anthony F. Graham, MD
Robert J. Howard, MD
Stuart Hutchison, MD
Victoria Korley, MD
Michael Kutryk, MD
Anatoly Langer, MD
Howard Leong-Poi, MD
Iqwal Mangat, MD
Gordon W. Moe, MD (Editor)
Juan C. Monge, MD (Assoc. Editor)
Thomas Parker, MD (Head)
Arnold Pinter, MD
Trevor I. Robinson, MD
Duncan J. Stewart, MD
Andrew Yan, MD

St. Michael's Hospital
30 Bond St.,
Suite 7049, Queen Wing
Toronto, Ont. M5B 1W8
Fax: (416) 864-5941

The opinions expressed in this publication do not necessarily represent those of the Division of Cardiology, St. Michael's Hospital, the University of Toronto, the educational sponsor, or the publisher, but rather are those of the author based on the available scientific literature. The author has been required to disclose any potential conflicts of interest relative to the content of this publication. *Cardiology Rounds* is made possible by an unrestricted educational grant.

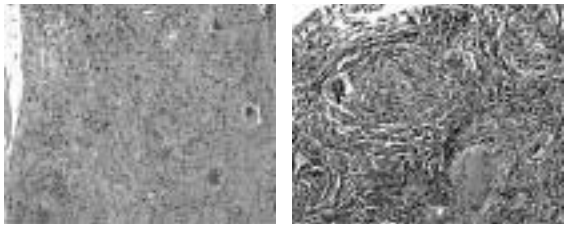
Leading with Innovation
Serving with Compassion

ST. MICHAEL'S HOSPITAL
A teaching hospital affiliated with the University of Toronto

Terrence Donnelly Heart Centre

UNIVERSITY OF TORONTO

Figure 1: Samples of myocardium involved with sarcoidosis.
A: Low power microscopy showing noncaseating, multinucleated giant cell granuloma.
B: Trichrome stain was used to show a dense band of collagen fibres, encasing aggregates of granulomas and inflammatory cells.



A B

Conduction abnormalities

Complete heart block is the most common finding in patients with clinically evident cardiac sarcoidosis. Clinical manifestations depend on the location and extent of granulomatous inflammation within the myocardium. First-degree heart block also occurs often, due to inflammation around the atrioventricular (AV) node or bundle of His.

Ventricular arrhythmias

Premature ventricular contractions and nonsustained ventricular tachycardia are common and observed with electrocardiograms (ECGs) in as many as 22% of patients with sarcoidosis.[8] Sudden death from ventricular tachyarrhythmias and complete heart block account for 25% to 65% of deaths from cardiac sarcoidosis.[9,10]

Heart failure

Heart failure can occur from extensive granulomatous infiltration of the myocardium with the possibility of both systolic and diastolic dysfunction. Occasionally, patients with extensive involvement of the left ventricular myocardium develop left ventricular aneurysms. Corticosteroid treatment may convert granulomas to scar tissue and contribute to the development of ventricular aneurysmal dilatation.[6] Progressive heart failure accounts for 25% to 75% of cardiac deaths from sarcoidosis.[9,10]

Supraventricular arrhythmias

These arrhythmias are infrequent and secondary to granulomatous involvement of the sinus node. This involvement can, for example, lead to ectopic atrial activity, paroxysmal atrial tachycardia, atrial flutter, atrial fibrillation, and sinus arrest.

Valvular dysfunction

Mitral incompetence due to papillary muscle involvement by sarcoid granulomas is usually transient; however, it may be severe enough to cause pulmonary hypertension and hemodynamic instability. Rarely, sarcoidosis involves the aortic valve and root, resulting in aortic regurgitation and aneurysm formation.

Simulated myocardial infarction (MI)

Cardiac sarcoidosis can produce clinical and ECG findings, including chest pain and pathologic Q waves that mimic MI. Occasionally, the localized granulomatous inflammation may mimic a transmural MI on diagnostic studies and gross pathologic examination.[11]

Extracardiac manifestations

Sarcoidosis usually presents with bilateral hilar adenopathy, pulmonary infiltrates, skin or eye lesions, or a combination of these abnormalities. The patient's immunologic and genetic background play a role, and symptoms vary by sex, race, and age at presentation:

- African Americans are more likely to have acute and severe disease
- women are more likely to have neurologic and ocular involvement
- men more frequently have abnormalities of calcium homeostasis.[12]

Sarcoidosis affects many systems and the sites involved other than the heart include the following.

The lungs are involved in 95% of patients. The most common initial complaints are cough, shortness of breath, and chest pain, often accompanied by fatigue, weakness, and malaise. In approximately one-half of patients, the disease is detected incidentally on chest radiographs before symptoms develop.[12]

The skin is involved in >20% of patients. Two types of lesions are common: lupus pernio (large bluish-red lesions on the nose, cheeks, ears, fingers, and toes) and erythema nodosum (raised, red, tender nodules on the front of the legs with occasional adjacent joint pain and swelling). Erythema nodosum is the hallmark of acute sarcoidosis.[13]

The eyes are involved in about 12% of patients. Any part of the eye may be affected, but uveitis is the most common. Acute anterior uveitis usually clears spontaneously, or after treatment with local steroid eye drops. Chronic uveitis forms adhesions between the lens and iris, causing glaucoma and blindness.[14]

Parotid and salivary glands are swollen in 5% of patients.[12] Peripheral lymph nodes are enlarged in about one-third of patients.

Abnormalities of calcium metabolism occur in 4% to 10% of patients[15] and are the most common electrolyte abnormality of sarcoidosis. Activated macrophages within the granulomas produce calcitriol, causing increased intestinal absorption of calcium, hypercalciuria and, eventually,

Table 1: Guidelines for diagnosing cardiac sarcoidosis[16]

1. Histologic diagnosis group

Cardiac sarcoidosis is confirmed when histologic analysis of operative or endomyocardial biopsy specimens demonstrates epithelioid granuloma without caseating granuloma.

2. Clinical diagnosis group

In patients with a histologic diagnosis of extracardiac sarcoidosis, cardiac sarcoidosis is suspected when item (a) and one or more of items (b) through (e) are present.

(a) Complete right bundle branch block, left axis deviation, atrioventricular block, VT, premature ventricular contraction (>Lawn 2), or abnormal Q or ST-T change on the ECG or ambulatory ECG.

(b) Abnormal wall motion, regional wall thinning, or dilatation of the left ventricle.

(c) Perfusion defect by thallium-201 myocardial scintigraphy or abnormal accumulation by gallium-67 or technetium-99m myocardial scintigraphy.

(d) Abnormal intracardiac pressure, low cardiac output, or abnormal wall motion or depressed ejection fraction of the left ventricle.

(e) Interstitial fibrosis or cellular infiltration over moderate grade even if the findings are nonspecific.

VT - ventricular tachycardia

hypercalcemia and nephrocalcinosis. Untreated calcium deposition within the kidneys can lead to chronic renal insufficiency and end-stage renal disease.[15]

Diagnosis

Cardiac involvement in sarcoidosis is extremely difficult to diagnose clinically because the clinical manifestations are non-specific, and the sensitivity and specificity of diagnostic modalities are limited. In 1993, the Japanese Ministry of Health and Welfare published guidelines for diagnosing cardiac sarcoidosis (Table 1).[16] However, these guidelines have not been rigorously validated in establishing the diagnosis of cardiac sarcoidosis. Diagnostic tests such as endomyocardial biopsy or imaging may be required, particularly in patients with no other manifestations of the disease.

Endomyocardial biopsy

False negative studies due to sampling error may occur because myocardial involvement is not homogeneous. Furthermore, myocardial sarcoid granulomas tend to be basal, while endomyocardial biopsy specimens are usually obtained from the apical septum. Sensitivity was 20% in one series of 26 patients.[17] Early endomyocardial biopsy is recommended whenever the diagnosis of cardiac sarcoidosis is considered. Although a negative biopsy does not exclude the diagnosis, the finding of noncaseating granulomas is diagnostic in the proper clinical setting (Figure 1).

Electrocardiography

ECG abnormalities are found in 70% of patients with sarcoidosis.[1] Holter monitoring can document and define subclinical rhythm disturbances that are missed on ECG. It has been recommended that 24-hour Holter monitoring and exercise electrocardiography be routinely performed in patients with suspected or known cardiac sarcoidosis.

Echocardiography

Sonography may display the sequelae of cardiac sarcoidosis such as ventricular aneurysms, valvular regurgitation, mitral valve prolapse, left ventricular dilatation, or segmental or global hypokinesia of the left ventricle. Cardiac tissue, particularly the ventricular septum or the left ventricular free wall, appears hyperechogenic when there is granulomatous involvement and scar formation.[18]

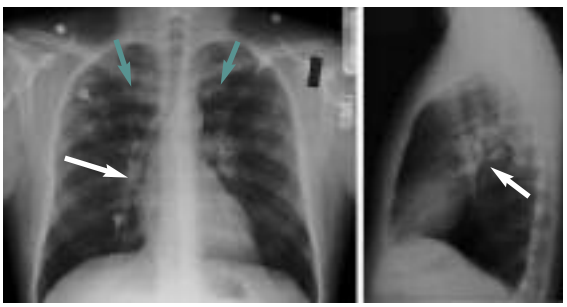
Chest radiography

Chest radiography may reveal cardiomegaly, congestive heart failure, pericardial effusion, or a left ventricular aneurysm. Findings consistent with pulmonary sarcoidosis, including hilar adenopathy, interstitial infiltrates, and honeycombing may also be noted (Figure 2).

Radionuclide imaging

Myocardial imaging with thallium-201 in patients with suspected cardiac sarcoidosis is useful to suggest myocardial involvement and to exclude cardiac dysfunction secondary to coronary artery disease (CAD). Rest

Figure 2: Anterior-posterior and lateral chest radiograph demonstrates hilar prominence (white arrows) and apical infiltrates (green arrows).



imaging may mimic patterns seen with CAD but, in contrast to CAD, the perfusion defects decrease in size during exercise for patients with sarcoidosis (reverse distribution). The combined use of thallium-201 and gallium-67 may improve the detection of cardiac sarcoidosis. Small series suggest that the presence of gallium-avid lesions may predict a greater response to corticosteroid treatment.[19] Fasting 18F-fluoro-2-deoxyglucose positron emission tomography (18F-FDG PET) can detect early inflammation from cardiac sarcoidosis, prior to the development of advanced fibrosis.[20]

Magnetic resonance imaging

Experience with magnetic resonance imaging (MRI) to diagnose or monitor myocardial sarcoidosis is more limited. Gadolinium-diethylenetriamine pentaacetic acid (DTPA) enhancement permits earlier detection of cardiac involvement and assessment of the efficacy of steroid therapy (Figure 3).[5] In one series, the sensitivity and specificity of late gadolinium enhancement were 100% and 78%, respectively.[21] Contrast-enhanced MRI and18F-FDG PET are the most sensitive and findings appear to correlate with disease activity.

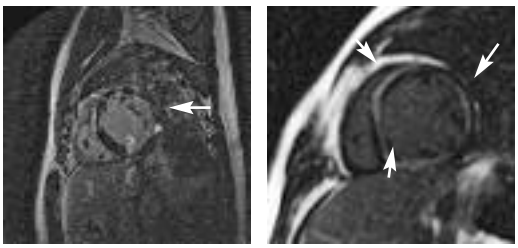
Coronary angiography

Coronary arteriography may be important in excluding the diagnosis of CAD. Perfusion defects on thallium-201 imaging in patients with known systemic sarcoidosis strongly suggest cardiac involvement if coronary angiography has excluded significant atherosclerosis. Vascular filling defects may be seen due to granulomas in the myocardium, wall motion abnormalities, and aneurysm formation may be seen on the ventriculogram.

Prognosis

The prognosis of patients afflicted with sarcoid heart disease has not been well-defined. An early necropsy series of 113 patients concluded that survival in most patients with symptomatic cardiac sarcoidosis was limited to about 2 years.[10] Substantially better outcomes were noted in later studies where 5-year survival was 40%-60%.[9,10] Whether the improvements in prognosis were due to early disease recognition (lead-time bias) or a relatively milder form of cardiac sarcoidosis versus early institution of corticosteroid treatment remain to be determined.

Figure 3: Examples of gadolinium-DTPA enhanced MRI in the detection of cardiac sarcoidosis



Treatment

Corticosteroids

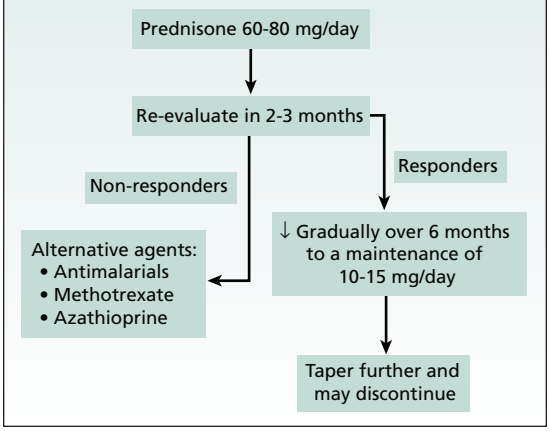
Corticosteroids are believed to be capable of halting or slowing the progression of inflammation and fibrosis in sarcoidosis; however, the data supporting efficacy are largely anecdotal. In a retrospective, survey-based study in 104 patients with cardiac sarcoidosis, survival was better with corticosteroid treatment than with usual care (64% versus 40%).[22] In another report, 75 patients with cardiac sarcoidosis treated with corticosteroids were compared with 20 patients not treated with corticosteroids in whom the diagnoses were proven at autopsy.[9] Five-year survival was much higher in steroid-treated patients (75% versus 10%). Among the treated patients, the outcome was best (89% 5-year survival) if steroids were begun when the left ventricle ejection fraction (LVEF) was 50%.

Treatment does not appear to reduce the incidence of ventricular arrhythmias. Generally, doses of 60 mg to 80 mg of prednisone daily are prescribed initially. Patients should be re-evaluated after 2 to 3 months and, if the disease is responding, the dose is tapered gradually to a maintenance level of 10 to 15 mg/day over a period of 6 months. If serial evaluations reveal that the disease is controlled, corticosteroids may be tapered further and eventually discontinued. Prerequisites for steroid taper or withdrawal include absence of disease activity confirmed by radionuclide or cardiac MRI, and by serum determinations of angiotensin-converting enzyme if values were initially elevated at time of diagnosis. Alternative agents such as antimalarials, methotrexate, and azathioprine may be given to patients who do not respond to corticosteroids or who cannot tolerate their side effects (Figure 4).[23] However, data supporting the efficacy of these alternative agents are largely anecdotal.

Antiarrhythmic treatment

Antiarrhythmic treatment and β-blockers are also often needed in the management of sarcoid

Figure 4: Suggested treatment algorithm for cardiac sarcoidosis[23]



heart disease. There have been no prospective studies evaluating the use of these agents in patients with cardiac sarcoidosis. Beta-blockers may actually increase the risk of heart block and amiodarone could exacerbate restrictive lung disease in sarcoidosis. Therefore, the physician must carefully weigh the benefits versus the risks of prescribing these medications.

Pacing and implantable cardiodefibrillators

Among patients with cardiac sarcoidosis, sudden death due to ventricular tachyarrhythmia or conduction block accounts for 30% to 65% of deaths.[6] There is a high rate of ventricular tachycardia recurrence or sudden death with antiarrhythmic drug therapy, even when guided by electrophysiologic testing. These observations constitute the rationale for the use of pacemakers and implantable cardiodefibrillators (ICDs). Permanent pacing is indicated when there is evidence of complete AV block or another high-grade conduction system disease is present. There is limited experience using ICDs in patients with cardiac sarcoidosis. It is likely that the main indications are similar to those in patients with other forms of cardiomyopathy.

Surgery and transplantation

Surgery is occasionally required to correct mitral valve disease or to resect a ventricular aneurysm for abolishing ventricular arrhythmias. Cardiac transplantation is an option for younger patients. Sarcoidosis is a systemic disease and sarcoid lesions in transplanted cardiac allografts can occur.[24]

Conclusion

Cardiac sarcoidosis can be a benign, an incidentally discovered condition, or a life-threatening disorder. Clinicians should consider the possibility of sarcoid heart disease in the evaluation of an otherwise healthy young or middle-aged person with cardiac symptoms, or in a patient with known sarcoidosis who develops arrhythmias, conduction disease, or heart failure.

References

1. Chapelon-Abric C, de Zuttere D, Duhaut P, et al. Cardiac sarcoidosis: a retrospective study of 41 cases. *Medicine (Baltimore)* 2004;83:315-334.
2. Thomsen TK, Eriksson T. Myocardial sarcoidosis in forensic medicine. *Am J Forensic Med Pathol* 1999;20:52-56.
3. Rybicki BA, Major M, Popovich J Jr, et al. Racial differences in sarcoidosis incidence: a 5-year study in a health maintenance organization. *Am J Epidemiol* 1997;145:234-241.
4. Silverman KJ, Hutchins GM, Bulkley BH. Cardiac sarcoid: a clinicopathologic study of 84 unselected patients with systemic sarcoidosis. *Circulation* 1978;58:1204-1211.
5. Shimada T, Shimada K, Sakane T, et al. Diagnosis of cardiac sarcoidosis and evaluation of the effects of steroid therapy by gadolinium-DTPA-enhanced magnetic resonance imaging. *Am J Med* 2001;110:520-527.
6. Roberts WC, McAllister HA, Ferrans VJ. Sarcoidosis of the heart. A clinicopathologic study of 35 necropsy patients (group 1) and review of 78 previously described necropsy patients (group 11). *Am J Med* 1977;63:86-108.
7. Ferrans VJ, Hibbs RG, Black WC, et al. Myocardial degeneration in cardiac sarcoidosis: histochemical and electron microscopic studies. *Am Heart J* 1965;69:159–72.
8. Sekiguchi M, Numao Y, Imai M. Clinical and histological profile of sarcoidosis for the heart and acute idiopathic myocarditis. Concepts through a study employing myocardial biopsy. I. Sarcoidosis. *Jpn Circ J* 1980;44:249-263.
9. Fleming HA, Bailey SM. The prognosis of sarcoid heart disease in the United Kingdom. *Ann N Y Acad Sci* 1986;465:543-550.
10. Yazaki Y, Isobe M, Hiroe M, et al. Prognostic determinants of long-term survival in Japanese patients with cardiac sarcoidosis treated with prednisone. *Am J Cardiol* 2001;88:1006-1110.
11. Wait J, Movahed A. Anginal chest pain in sarcoidosis. *Thorax* 1989;44:391-395.
12. Baughman RP, Teirstein AS, Judson MA, et al. Clinical characteristics of patients in a case control study of sarcoidosis. *Am J Respir Crit Care Med* 2001;164:1885-1889.
13. Lofgren S. Erythema nodosum studies on etiology and pathogenesis in 185 adult cases. *Acta Med Scand* 1946;suppl 174:1-197.
14. Karma A. Ophthalmic changes in sarcoidosis. *Acta Ophthalmol* 1979;141(suppl):1-94.
15. Rizzato G, Colombo P. Nephrolithiasis as a presenting feature of chronic sarcoidosis: a prospective study. *Sarcoidosis Vasc Diffuse Lung Dis* 1996;13:167-172.
16. Hiraga H, Yuwai K, Hiroe M, et al. Guideline for diagnosis of cardiac sarcoidosis: study report on diffuse pulmonary diseases from the Japanese Ministry of Health and Welfare. Tokyo: Japanese Ministry of Health and Welfare. 1993:23-24.
17. Uemura A, Morimoto S, Hiramitsu S, Kato Y, Ito T, Hishida H. Histologic diagnostic rate of cardiac sarcoidosis: evaluation of endomyocardial biopsies. *Am Heart J* 1999;138:299-302.
18. Fahy CJ, Marwick T, McGreery CJ, Quigley PJ, Maurer BJ. Doppler echocardiography detection of left ventricular diastolic dysfunction in patients with pulmonary sarcoidosis. *Chest* 1996;109:62-66.
19. Okayama K, Kurata C, Tawarahara K, et al. Diagnostic and prognostic value of myocardial scintigraphy with thallium-201 and gallium-67 in cardiac sarcoidosis. *Chest* 1995;107:330-334.
20. Okumura W, Iwasaki T, Toyama T, et al. Usefulness of fasting 18F-FDG PET in identification of cardiac sarcoidosis. *J Nucl Med* 2004;45:1989-1998.
21. Smedema JP, Snoep G, van Kroonenburgh MP, et al. Evaluation of the accuracy of gadolinium-enhanced cardiovascular magnetic resonance in the diagnosis of cardiac sarcoidosis. *J Am Coll Cardiol* 2005;45:1683-1690.

22. Takada K, Ina Y, Yamamoto M, Satoh T, Morishita M. Prognosis after pacemaker implantation in cardiac sarcoidosis in Japan. Clinical evaluation of corticosteroid therapy. *Sarcoidosis* 1994;11:113-117.
23. Doughan AR, Williams BR. Cardiac sarcoidosis. *Heart* 2006;92:282-288.
24. Oni AA, Hershberger RE, Norman DJ, et al. Recurrence of sarcoidosis in a cardiac allograft: control with augmented corticosteroids. *J Heart Lung Transplant* 1992;11:367-369.

Abstracts of Interest

Evaluation of the accuracy of gadolinium-enhanced cardiovascular magnetic resonance in the diagnosis of cardiac sarcoidosis.

SMEDEMA JP, SNOEP G, VAN KROONENBURGH MP, VAN GEUNS RJ, DASSEN WR, GORGELS AP, CRIJNS HJ.

OBJECTIVES: This study analyzed the accuracy of gadolinium-enhanced cardiovascular magnetic resonance (CMR) for the diagnosis of cardiac sarcoidosis (CS).

BACKGROUND: The diagnosis of CS was made according to the guidelines of the Japanese Ministry of Health and Welfare (1993); CMR has not been incorporated into the guidelines, and the diagnostic accuracy of CMR for the diagnosis of CS has not yet been evaluated.

METHODS: We performed an analysis of 12-lead electrocardiograms (ECGs), 24-h ambulatory ECGs, echocardiograms, thallium scintigrams, and gadolinium-enhanced CMR studies in 58 biopsy-proven pulmonary sarcoidosis patients assessed for CS. The diagnostic accuracy of CMR for CS was determined using modified Japanese guidelines as the gold standard.

RESULTS: The diagnosis of CS was made in 12 of 58 patients (21%); CMR revealed late gadolinium enhancement (LGE), mostly involving basal and lateral segments (73%), in 19 patients. In 8 of the 19 patients, scintigraphy was normal, while patchy LGE was present. The sensitivity and specificity of CMR were 100% (95% confidence interval, 78% to 100%) and 78% (95% confidence interval, 64% to 89%), and the positive and negative predictive values were 55% and 100%, respectively, with an overall accuracy of 83%.

CONCLUSIONS: In patients with sarcoidosis, CMR is a useful diagnostic tool to determine cardiac involvement. New diagnostic guidelines should include CMR.

J Am Coll Cardiol 2005;45(10):1683-90.

Prognostic determinants of long-term survival in Japanese patients with cardiac sarcoidosis treated with prednisone.

YAZAKI Y, ISOBE M, HIROE M, MORIMOTO S, HIRAMITSU S, NAKANO T, IZUMI T, SEKIGUCHI M.

Cardiac involvement is an important prognostic factor in sarcoidosis, but reliable indicators of mortality risk in cardiac sarcoidosis are unstudied in a large number of patients. To determine the significant predictors of mortality and to assess the efficacy of corticosteroids, we analyzed clinical findings, treatment, and prognosis in 95 Japanese patients with cardiac sarcoidosis. Twenty of these 95 patients had cardiac sarcoidosis proven by autopsy; none of these patients had received corticosteroids. We assessed 12 clinical variables as possible predictors of mortality by Cox proportional hazards model in 75 steroid-treated patients. During the mean follow-up of 68 months, 29 patients (73%) died of congestive heart failure and 11 (27%) experienced sudden death. Kaplan-Meier survival curves showed 5-year survival rates of 75% in the steroid-treated patients and of 89% in patients with a left ventricular ejection fraction > or = 50%, whereas there was only 10% 5-year survival rate in autopsy subjects. There was no significant difference in survival curves of patients treated with a high initial dose (> 30 mg) and a low initial dose (> or = 30 mg) of prednisone. Multivariate analysis identified New York Heart Association functional class (hazard ratio 7.72 per class I increase, p = 0.0008), left ventricular end-diastolic diameter (hazard ratio 2.60/10 mm increase, p = 0.02), and sustained ventricular tachycardia (hazard ratio 7.20, p = 0.03) as independent predictors of mortality. In conclusion, the severity of heart failure was one of the most significant independent predictors of mortality for cardiac sarcoidosis. Starting corticosteroids before the occurrence of systolic dysfunction resulted in an excellent clinical outcome. A high initial dose of prednisone may not be essential for treatment of cardiac sarcoidosis.

Am J Cardiol 2001;88(9):1006-10.

Upcoming meetings

1-5 September 2007
European Society of Cardiology Annual Meeting
Vienna, Austria
Contact: www.escardio.org

20-24 October 2007
Canadian Cardiovascular Society CCC 2007
Quebec City, Quebec
Contact: www.cardiocongress.org

Disclosure Statement: *Dr. Moe and Dr. Baslaib have declared that they have no conflicts of interest to disclose in association with the contents of this issue.*

Change of address notices and requests for subscriptions to *Cardiology Rounds* are to be sent by mail to P.O. Box 310, Station H, Montreal, Quebec H3G 2K8 or by fax to (514) 932-5114 or by e-mail to info@snellmedical.com. Please reference *Cardiology Rounds* in your correspondence. Undeliverable copies are to be sent to the address above. Publications Post #40032303

This publication is made possible by an educational grant from

Novartis Pharmaceuticals Canada Inc.

© 2007 Division of Cardiology, St. Michael's Hospital, University of Toronto, which is solely responsible for the contents. Publisher: **SNELL Medical Communication Inc.** in cooperation with the Division of Cardiology, St. Michael's Hospital, University of Toronto. ®*Cardiology Rounds* is a registered trademark of SNELL Medical Communication Inc. All rights reserved. The administration of any therapies discussed or referred to in *Cardiology Rounds* should always be consistent with the approved prescribing information in Canada. **SNELL Medical Communication Inc.** is committed to the development of superior Continuing Medical Education.