Cardiovascular Reviews, 2023, Volume 1, Issue 1
http://ojs.bbwpublisher.com/index.php/CR

Importance of Cardiac Rehabilitation in Patients with Structural Heart Changes Secondary to Ankylosing Spondylitis: A Case Report

Myllena Lemes Scardino Vieira*, Tamires Mota Toni, Claudio Spinola Najas

University of the West of São Paulo (UNOESTE), Presidente Prudente, SP, Brazil

****Corresponding author:*** Myllena Lemes Scardino Vieira, myllenalemes15@gmail.com

Abstract: Patients with ankylosing spondylitis (AS) may present with cardiac manifestations, mainly valvular disease and dilation of the aortic root. Rehabilitation programs to improve cardiovascular capacity are important for maintaining the quality of life of these patients, which often deteriorates with disease impairments and dysfunctions when not corrected. We report a case of a 67-year-old man, diagnosed with ankylosing spondylitis 20 years ago with structural cardiac changes. The patient underwent a rehabilitation program that involves stretching, aerobic and resistance exercises, and health education. In order to analyze the patient's effort during activities of daily living and instrumental activities, we used Borg Rating of Perceived Exertion Scale. With constant rehabilitation, the patient maintained functional capacity in activities of daily living and instrumental activities over the years even with structural heart changes.

Keywords: Heart disease; Heart valves; Ankylosing spondylitis; Connective tissue

Online publication: May 22, 2023

1. Introduction

Ankylosing spondylitis (AS) is considered a chronic disease that directly affects the connective tissue, axial skeleton, spine, and sacroiliac joints, resulting in their fusion. Patients with AS commonly present with cardiac manifestations, such as aortic root dilatation, valve disease, conduction and rhythm abnormalities, decreased coronary flow reserve, myocardial infarction, and diastolic dysfunction [1,2].

The main clinical manifestations of AS include back pain and progressive stiffness of the spine, as well as inflammation of the hip joint, shoulders, peripheral joints, fingers, and toes [3]. In addition to physical changes, studies have shown that the onset of comorbidities in AS intensifies the progression of the disease, contributing to the onset of functional disability and increased mortality [4].

Among the comorbidities caused by AS, cardiac manifestations, such as aortic disease and aortic regurgitation [5], have been studied. Based on the National Health Insurance Research Database of Taiwan, a cohort study conducted between 2000 and 2005 demonstrated a relationship between AS and cardiac manifestations and the prevalence. A significant number of cardiac complications has been identified in male patients over 40 years old and diagnosed with AS [2].

Other studies have revealed a prevalence of cardiac complications of 0.1%–0.5% in patients with AS, predominantly in the Caucasian population, in young HLA-B27 positive males (female-to-male ratio 1:9), and in those with spinal and sacroiliac join involvement [6,7].

The implementation of rehabilitation programs with physical activity improves the cardiovascular capacity and quality of life of patients with cardiac manifestations secondary to AS [8]. Regular physical exercise allows physiological restructuring of the cardiovascular and musculoskeletal systems, reducing oxidative stress and inflammation, correcting baroreflex dysfunction, increasing vagal tone, decreasing sympathetic activity, reversing hypertrophic arteriolar remodeling in exercised tissues, and reducing peripheral vascular resistance (reducing BP and controlling similar blood pressure levels) [8-10]. Thus, it is necessary to evaluate the clinical changes in these patients after rehabilitation.

In this study, we evaluated the benefits of rehabilitation in a patient with structural heart changes complicated by AS who showed maintenance of functional capacity after cardiac rehabilitation.

2. Case

A 67-year-old male, former smoker, with non-insulin-dependent diabetes mellitus, systemic arterial hypertension, AS, and heart failure with reduced ejection fraction, followed-up at the cardiac rehabilitation center of a physiotherapy clinic in the West of São Paulo. The patient signed an informed consent form, agreeing to participate in the study. This study was approved by the Research Ethics Committee of Universidade do Oeste Paulista (CAAE: 51309321.4.0000.5515), in accordance with CNS Resolution 466/2012.

In 2001, the patient compliant of severe pain in the cervical and lumbar region, and he was diagnosed with AS. In 2013, he had an episode of rheumatic fever, with consequent involvement of the cardiovascular system. The echocardiogram at the time revealed mild mitral valve insufficiency and stenosis due to calcification in the mitral valve. Since then, the patient had been undergoing cardiovascular rehabilitation. In 2016, his cardiovascular condition worsened, with episodes of shortness of breath. He underwent 24-hour Holter monitoring, which identified a variation in heart rate from 49 to 176 beats per minute and four episodes of non-sustained ventricular tachycardia. In 2017, his echocardiogram showed left ventricular contractile dysfunction (ejection fraction of 46.6%), mitral dysfunction with stenosis and insufficiency, significant left atrial enlargement, and mild dilatation of the ascending aorta. Due to the clinical picture and structural cardiac changes, a pacemaker was implanted in the same year. Then, the patient was placed under the cardiac rehabilitation protocol; however, in 2018, he presented with atrial fibrillation, as shown on electrocardiogram.

Another echocardiogram was performed in 2020, showing irregular rhythm, significant left ventricular contractile dysfunction (ejection fraction of 24.4%), mitral dysfunction with moderate stenosis and insufficiency, mild aortic insufficiency, mild/moderate tricuspid insufficiency, and significant left atrial enlargement.

Under the cardiac rehabilitation program, the patient was classified as high risk according to the American College of Sports Medicine (ACSM) in view of cardiovascular disease (rheumatic valvulopathy and heart failure with reduced ejection fraction) and metabolic disease (diabetes mellitus). The Cardiovascular Risk Stratification chart (**Table 1**) was used to stratify the patient’s risk, and heart rate reserve (HRR) was used to calculate the intensity of effort, in the range of 40%–70%, as recommended by the ACSM. The heart rate reserve ranged from 103 to 119 beats per minute.

The patient was classified as high risk under cardiac rehabilitation and as class 3 by the New York Heart Association (NYHA) classification of heart failure based on his symptoms in the last evaluation. The rehabilitation program involved stretching exercises, aerobic exercises, resistance exercises, and health education. The program began with self-stretching exercises of the main muscle groups (neck, trunk, and upper and lower limbs), maintaining over 30 seconds in standing or sitting position. Aerobic exercises were performed on a treadmill, beginning with a 5-minute warm-up, and then 20 minutes of moderate intensity physical conditioning, calculated by means of HRR and Borg Rating of Perceived Exertion Scale. At the end of the aerobic activity, 5 minutes was spent to cool down. Resistance exercises were performed at the clinic's gym, and for each exercise, the ideal load was calculated based on the patient's maximum repetition (1RM) according to his body percentage. In order to analyze the subjective effort of the patient during aerobic exercises and activities of daily living, we used Borg Rating of Perceived Exertion Scale (**Table 2**).

Table 1. American College of Sports Medicine risk stratification chart

Risk	Criteria
Low	Men under 45 years of age and women under 55 years of age who are asymptomatic and do not meet more than one major risk (positive factors: family history, smoking, hypertension, hypercholesterolemia, increased fasting glucose, obesity, and sedentary lifestyle; negative factors: high serum HDL cholesterol).
Moderate	Men aged 45 or older, women aged 55 or older, or those who meet the threshold for two or more major risk factors described above.
High	Individuals with one or more signs and symptoms (pain; discomfort in the chest, neck, jaw, or arms; shortness of breath at rest or on exertion; dizziness or syncope; orthopnea or paroxysmal nocturnal dyspnea; swelling of the ankles; palpitations or tachycardia; intermittent claudication; known heart murmur; excessive fatigue; and shortness of breath in daily activities) or cardiovascular disease (heart disease, cerebrovascular disease, and peripheral vascular disease), lung disease (chronic obstructive pulmonary disease, asthma, interstitial lung disease, and cystic fibrosis), or known metabolic disease (diabetes mellitus, thyroid disorders, and kidney or liver disease).

Table 2. Borg Rating of Perceived Exertion

6 7 8	Very easy
9 10	Easy
11 12	Relatively easy
13 14	Relatively tiring
15 16	Tiring
17 18	Very tiring
19 20	Exhaustive

3. Discussion

Patients with AS are more susceptible to developing aortic insufficiency, conduction abnormalities, aortitis, pericarditis, and even mitral insufficiency, as observed by the classic study by Kawasuji *et al*. [11]. Of these changes, aortic disease and aortic regurgitation are the most common cardiovascular diseases associated with AS [12].

Since that study, several reports describing these associations have been published. Thomas *et al*. [13] observed that among the AS patients studied, 27% of them had mild aortic root dilatation and one of them aortic insufficiency. Roldan *et al*. [14] reported that the prevalence of valve disease was 82% in patients with AS.

This information corroborates the findings in our study, in which the patient developed aortic and mitral insufficiency, in addition to aortic dilatation. Although he presented with rheumatic fever, as a triggering factor, these structural cardiac alterations may have been accentuated by AS. However, it is impossible to determine the exact mechanism of the cause-and-effect relationship between rheumatic fever/AS and heart disease [15].

Even with the worsening structural cardiac parameters in the 2017 to 2020 echocardiograms, a progression that is part of the natural history of rheumatic fever/heart failure, the patient maintained his quality of life and functional independence through cardiac rehabilitation as demonstrated by the NYHA scale and functional capacity assessment [15,16].

SPSS 22.0 was used for statistical analysis. Data normality was assessed by Kolmogorov-Smirnov test. For comparison of parametric data, independent *t*-test was used, while for non-parametric data, Mann-Whitney test was used. The data were described in mean ± standard deviation. The significance level considered was 5%.

In the case study, the patient's vital signs at rest and during exercise were observed. **Tables 1**–**7** detail the averages of the vital signs at each of the times.

In the tables, Time 1 and Time 2 refer to 2018 and 2021, respectively. They are arranged according to the collection time, as the number of sessions varied with each time, and for this reason, each table will have the number of sessions (n).

Table 1. Vital signs during rest

	Time 1 (n = 29)	**Time 2 (n = 22)**	***P***
SBP	105.52 ± 10.21	103.64 ± 13.99	0.5810
DBP	69.62 ± 5.66	70.00 ± 8.16	0.8455
HR	70.90 ± 11.52	66.77 ± 11.16	0.2054
$SatO_2$	96.45 ± 2.49	97.18 ± 0.59	0.1826
Borg	8.03 ± 2.03	7.21 ± 1.90	0.1607

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; $SatO_2$, peripheral oxygen saturation; SBP, systolic blood pressure.

At rest, no significant statistical differences in vital signs were observed in the two "times" analyzed. **Tables 2**–**7** show a significant reduction in heart rate and subjective perceived exertion in Time 2 at the 5th, 10th, 15th, 20th, 25th, and 30th minutes of exercise.

Table 2. Vital signs at the 5th minute of exercise

	Time 1 (n = 29)	**Time 2 (n = 22)**	***P***
SBP	107.24 ± 12.51	105.91 ± 14.03	0.7222
DBP	69.31 ± 5.51	70.91 ± 8.68	0.2552
HR	85.83 ± 16.16	69.82 ± 12.16	0.0003*
$SatO_2$	96.14 ± 2.42	96.45 ± 1.60	0.5970
Borg	11.66 ± 1.14	9.41 ± 2.46	0.0001*

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; $SatO_2$, peripheral oxygen saturation; SBP, systolic blood pressure.
*$P < 0.005$.

Table 3. Vital signs at the 10th minute of exercise

	Time 1 (n = 29)	Time 2 (n = 22)	*P*
SBP	110.34 ± 12.67	111.43 ± 14.59	0.7806
DBP	69.62 ± 6.81	72.38 ± 8.89	0.2195
HR	94.21 ± 12.70	69.48 ± 12.09	0.0000*
$SatO_2$	95.66 ± 4.14	95.90 ± 2.47	0.8068
Borg	12.10 ± 0.72	10.43 ± 2.16	0.0003*

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; $SatO_2$, peripheral oxygen saturation; SBP, systolic blood pressure.
*$P < 0.005$.

Table 4. vital signs at the 15th minute of exercise

	Time 1 (n = 29)	Time 2 (n = 22)	*P*
SBP	111.00 ± 12.64	114.50 ± 14.32	0.3714
DBP	70.31 ± 6.81	72.00 ± 8.34	0.4400
HR	95.17 ± 14.81	71.70 ± 11.36	0.0000*
$SatO_2$	9634 ± 2.33	97.05 ± 1.43	0.2354
Borg	12.17 ± 0.76	10.85 ± 2.21	0.0044*

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; $SatO_2$, peripheral oxygen saturation; SBP, systolic blood pressure.
*$P < 0.005$.

Table 5. vital signs at the 20th minute of exercise

	Time 1 (n = 29)	Time 2 (n = 22)	*P*
SBP	112.14 ± 13.43	111.54 ± 12.14	0.8909
DBP	71.07 ± 6.29	69.23 ± 8.62	0.4438
HR	96.86 ± 13.07	74.85 ± 14.74	0.0000*
$SatO_2$	96.61 ± 2.44	96.46 ± 1.13	0.8392
Borg	12.36 ± 0.83	11.38 ± 1.98	0.0312*

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; $SatO_2$, peripheral oxygen saturation; SBP, systolic blood pressure.
*$P < 0.005$.

Table 6. Vital signs at the 25th minute of exercise

	Time 1 (n = 29)	Time 2 (n = 22)	*P*
SBP	112.48 ± 8.29	110.00 ± 8.16	0.5826
DBP	72.40 ± 5.97	70.00 ± 11.55	0.5190
HR	97.00 ± 13.04	75.25 ± 21.50	0.0085*
$SatO_2$	96.84 ± 1.97	96.50 ± 0.58	0.7382
Borg	12.48 ± 0.51	10.00 ± 1.83	0.0000*

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; $SatO_2$, peripheral oxygen saturation; SBP, systolic blood pressure.
*$P < 0.005$.

There was also a significant reduction in diastolic blood pressure in Time 2 at the 30th minute of exercise (**Table 7**).

Table 7. Vital signs at the 30th minute of exercise

	Time 1 (n = 29)	Time 2 (n = 22)	*P*
SBP	109.65 ± 7.69	100.00 ± 0.00	0.0951
DBP	72.17 ± 6.71	60.00 ± 0.00	0.0193
HR	98.00 ± 11.78	72.00 ± 16.97	0.0076*
$SatO_2$	96.91 ± 1.35	96.00 ± 1.41	0.3679
Borg	11.78 ± 1.65	9.00 ± 1.41	0.0308*

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; $SatO_2$, peripheral oxygen saturation; SBP, systolic blood pressure.
*$P < 0.005$.

As described in literature and reported in this case, AS can cause cardiac manifestations that are potentially detrimental to the quality of life of patients due to several factors. Cardiac rehabilitation has shown to be effective in delaying the progression of symptoms, even with structural changes in the heart, which in this case resulted in a significant decrease in the patient's left ventricular ejection fraction. The maintenance of functional capacity for activities of daily living through the subjective perception of effort and during exertion in cardiovascular rehabilitation was observed, as the patient only had physiological changes in their vital data throughout the sessions.

Disclosure statement

The authors declare that there is no potential conflict of interest that could interfere with the impartiality of this scientific work.

References

[1] Amin A, Chitsazan M, Navid H, 2016, Left Ventricular Systolic Dysfunction in Two Patients with Ankylosing Spondylitis: What Is the Role of Corticosteroids?. Eur J Rheumatol, 3(4): 179–181. https://dx.doi.org/10.5152/eurjrheum.2016.15069

[2] Hung YM, Chang WP, Wei JC, et al., 2016, Midlife Ankylosing Spondylitis Increases the Risk of Cardiovascular Diseases in Males 5 Years Later: A National Population-Based Study. Medicine, 95(18): e3596. https://dx.doi.org/10.1097/MD.0000000000003596

[3] Zhu W, He X, Cheng K, et al., 2019, Ankylosing Spondylitis: Etiology, Pathogenesis, and Treatments. Bone Res, 7: 22. https://dx.doi.org/10.1038/s41413-019-0057-8

[4] Rosenbaum JT, Pisenti L, Park Y, et al., 2019, Insight into the Quality of Life of Patients with Ankylosing Spondylitis: Real-World Data from a US-Based Life Impact Survey. Rheumatol Ther, 6(3): 353–367. https://dx.doi.org/10.1007/s40744-019-0160-8

[5] Grygiel-Górniak B, Oduah MT, Olagunju A, et al., 2020, Disorders of the Aorta and Aortic Valve in Connective Tissue Diseases. Curr Cardiol Rep, 22(8): 70. https://dx.doi.org/10.1007/s11886-020-01314-0

[6] Talotta R, Rucci F, Scaglione F, 2020, Calcium Physiology, Metabolism and Supplementation: A Glance at Patients with Ankylosing Spondylitis. Reumatologia, 58(5): 297–311. https://dx.doi.org/10.5114/reum.2020.100112

[7] Ward MM, Deodhar A, Gensler LS, et al., 2019, Update of the American College of Rheumatology/Spondylitis Association of America/Spondyloarthritis Research and Treatment Network Recommendations for the Treatment of Ankylosing Spondylitis and Nonradiographic Axial

Spondyloarthritis. Arthritis Rheumatol, 71(10): 1599–1613. https://dx.doi.org/10.1002/art.41042

[8] Masson GS, Michelini LC, 2015, Autonomic Dysfunction, Sympathetic Hyperactivity and the Development of End-Organ Damage In Hypertension: Multiple Benefits of Exercise Training. Heart Res Open J, 2(2): 60–69. https://dx.doi.org/10.17140/HROJ-2-111

[9] Herrod PJJ, Doleman B, Blackwell JEM, et al., 2018, Exercise and Other Nonpharmacological Strategies to Reduce Blood Pressure in Older Adults: A Systematic Review and Meta-Analysis. J Am Soc Hypertens, 12(4): 248–267. https://dx.doi.org/10.1016/j.jash.2018.01.008

[10] Araujo CAB, Constantinov KM, Azevedo MVGT, et al., 2015, Tratamento Fisioterapêutico em Espondilite Anquilosante: Uma Revisão Sistemática [Physiotherapy Treatment in Ankylosing Spondylitis: A Systematic Review]. RUEP, 12(29): 53–58.

[11] Kawasuji M, Hetzer R, Oelert H, et al., 1982, Aortic Valve Replacement and Ascending Aorta Replacement in Ankylosing Spondylitis: Report of Three Surgical Cases and Review of the Literature. Thorac Cardiovasc Surg, 30: 310–314. https://dx.doi.org/10.1055/s-2007-1022414

[12] Yuan SM, 2009, Cardiovascular Involvement of Ankylosing Spondylitis: Report of Three Cases. Vascular, 17(6): 342–354. https://dx.doi.org/10.2310/6670.2009.00023

[13] Thomas D, Hill W, Geddes R, et al., 1982, Early Detection of aortic Dilatation in Ankylosing Spondylitis Using Echocardiography. Aust N Z J Med, 12: 10–13. https://dx.doi.org/10.1111/j.1445-5994.1982.tb02416.x

[14] Roldan CA, Chavez J, Wiest PW, et al., 1998, Aortic Root Disease and Valve Disease Associated with Ankylosing Spondylitis. J Am Coll Cardiol, 32: 1397–1404. https://dx.doi.org/10.1016/s0735-1097(98)00393-3

[15] Figueiredo ET, Azevedo L, Rezende ML, et al., 2019, Rheumatic Fever: A Disease Without Color. Arq Bras Cardiol, 113(3): 345–354. https://doi.org/10.5935/abc.20190141

[16] Santos AB, Cruz WA, Pinho RPWS, et al., 2015, Monitoramento Da Carga Interna de Treinamento Através de Escalas de Percepção Subjetiva de Borg, Foster e Dor [Training Load Monitoring Through Borg, Foster and Pain Subjective Perception Scales. RBPFEX, 9(52): 121–128.