



Cardiomyopathy Presenting as Severe Fatigue in a Person With Chronic Spinal Cord Injury

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Abstract

Background/Objective: To describe a case of fatigue associated with cardiomyopathy in a man with spinal cord injury.

Study Design: Case report.

Subject: An obese 35-year-old man with long-term ASIA A L2 paraplegia, accompanied by a 2-year history of progressive severe fatigue.

Methods: Physical examination showed obesity, hypertension, tachycardia, and pitting edema. An echocardiogram showed a 20% ejection fraction, severe dilation, and global hypokinesis in the left ventricle and mild to moderate mitral regurgitation.

Results: Symptoms improved after treatment with furosemide, warfarin, ramipril, and continuous positive airway pressure for obstructive sleep apnea.

Conclusion: Severe progressive fatigue in a patient with chronic SCI may signal cardiomyopathy. Diagnostic studies may be warranted in patients with progressive fatigue.

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Key Words: Cardiomyopathy; Spinal cord injuries; Paraplegia; Congestive heart failure; Mitral regurgitation; Fatigue; Chronic obstructive pulmonary disease; Obstructive sleep apnea; Obesity

INTRODUCTION

Fatigue is a common symptom reported by patients with spinal cord injuries (SCIs) in the postacute and post-discharge phases (1). The prevalence of fatigue may be as high as 57% and may result in functional impairment (2). Intrinsic fatigue is the result of histologic and metabolic changes in complete or partially denervated muscles at the level of or below the lesion (3). In addition, contributing factors may include metabolic and endocrine disturbances, obesity, heart or respiratory failure, sleep apnea, pain from muscle overuse, rehospitalization, and difficulties coping with chronic injury (3–5) (Table 1). Even though heart failure is one cause of chronic fatigue in patients with chronic SCI, a paucity of research exists regarding the causes of heart failure in this population.

Currently, coronary artery disease (CAD) is a leading cause of morbidity and mortality in individuals with SCI

because of their elevated percentage of body fat and sedentary lifestyle (6–9). A study examining the prevalence of CAD in 47 clinically asymptomatic patients with chronic SCI also reported that those individuals with combined tetraplegia and complete injuries experienced an increased risk for CAD. In this study, 63.8% had abnormal results on cardiac stress testing with thallium-201 myocardial perfusion single photon emission computed tomography (TI-201 SPECT) (10).

The majority of human and animal research studies in the area of SCI and cardiovascular diseases are related to the acute phase of SCI, especially in individuals with higher levels of SCI (ie, cervical to mid-thoracic) (11–14). Individuals with cervical and/or upper thoracic spinal injuries tend to have cardiovascular instability, including unstable arterial pressure and heart rate. As a result, they are at a higher risk for hypertensive cardiovascular diseases (15). With respect to lower level SCI (ie, below T12), one animal study showed that a dog developed symptoms of neurogenic cardiomyopathy only a few days after an acute SCI in the lumbosacral region (16). Pahl et al (17) examined cardiovascular complications in 20 men (age range, 39–63 years) with SCI (levels of injury, C4–T12) of 6- to 32-year duration who were also on hemodialysis.

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Table 1. Differential Diagnosis of Fatigue After SCI^{3,32,33}

Categories	Differential Diagnosis
Postinjury	Intrinsic fatigue of paralyzed muscles Pain Obesity
Toxin	Medication side effects Alcohol or drug abuse
Infection	Viral syndromes Mononucleosis Hepatitis Endocarditis
Neoplastic conditions	Post-polio syndrome Leukemia Lymphoma Occult malignancy
Endocrine and metabolic problems	Electrolyte disorders Hypothyroidism Diabetes mellitus Addison's disease Malnutrition
Vascular disorders	Congestive heart failure Valvular heart disease Cardiomyopathies
Pulmonary conditions	Respiratory failure Chronic obstructive pulmonary disease Restrictive lung diseases
Sleep disorders	Sleep apnea Narcolepsy Restless legs syndrome Primary insomnia
Psychosocial problems	Depression Anxiety Adjustment reactions Situational life distress Sexual dysfunction Family violence Occupational stress Re-hospitalization Difficulties coping with the postinjured circumstance
Miscellaneous conditions	Professional burnout Anemia Pregnancy Connective tissue disease Fibromyalgia

Relevant postmortem data indicated that all subjects exhibited cardiovascular abnormalities: pericarditis (45%); left and right ventricular hypertrophy (45% and 20%); left and right ventricular dilation (40% and 30%); cardiac amyloidosis (25%); and myocardial fibrosis (45%). In this study, 1 case of aortic stenosis and 2 cases of mitral valve dilatation were reported, whereas no evidence of vegetation, endocarditis, or acute myocardial infarction was noted. The authors speculated that the high

prevalence of ventricular dilation was possibly from long-term elevated cardiac output secondary to chronic fluid overload in this population with end-stage renal disease.

PubMed/MEDLINE was searched, without limitations, in December 2007. Search terms included cardiomyopathy, spinal cord injury, fatigue, heart failure, obesity, dilated cardiomyopathy (DCM), spinal cord injury comorbidity, cardiac complication(s), sleep apnea, and myocardial changes. All pertinent literature was reviewed to identify other similar cases, but no cases of cardiomyopathy and long-term SCI were found.

CASE DESCRIPTION

A 35-year-old man with L2 ASIA A paraplegia presented with a history of fatigue severe enough to interfere with his ability to work full time. He was injured in a motor vehicle crash 19 years before admission, resulting in a fracture/dislocation of T12 to L1 with immediate paraplegia. With mobilization and increased activities, he reached independence at the wheelchair level. He sporadically attended follow-up appointments, citing lethargy, and his last appointment was 4 years ago. He was admitted to our rehabilitation hospital for further study.

On admission, he reported lethargy, daytime somnolence, and a tendency to snore excessively at night. A gradual gain in weight of 35 lb over 2 years was accompanied by a history of low affect over the previous 3 months and disinterest in daily activities and socialization. However, no recent psychosocial stressors could be identified. He also reported nocturnal panic attacks that began with shortness of breath and were associated with nausea, sweating, hot flushes, and tremor.

Medications before admission included Combivent inhaler (2 puffs; 4 times/d) and the NicoDerm patch. He was a 19-pack-year smoker and a social drinker. Complications since the injury included recurrent urinary tract infections, constipation, neurogenic bladder, neurogenic bowel, and erectile dysfunction. His history was significant for childhood asthma, which has been in remission since the age of 12 years. The patient was divorced and lived alone in a condominium apartment. Before admission, he had been employed as a seed analyst supervisor for a private company. He used a wheelchair on a full-time basis and he was independent with activities of daily living (ADLs) and instrumental activities of daily living (IADLs), except for requiring assistance with bathing. He reported that he had poor eating habits and often ordered take-out food.

Physical Examination

At admission, he was morbidly obese; body weight was 129 kg (body mass index [BMI], 40.8 kg/m²). He was alert, oriented, and in no apparent distress. Pulse was 124/min and regular; blood pressure was 160/102 mmHg. Jugular venous pulse was not raised, and heart sounds were normal with no audible murmurs. Pitting

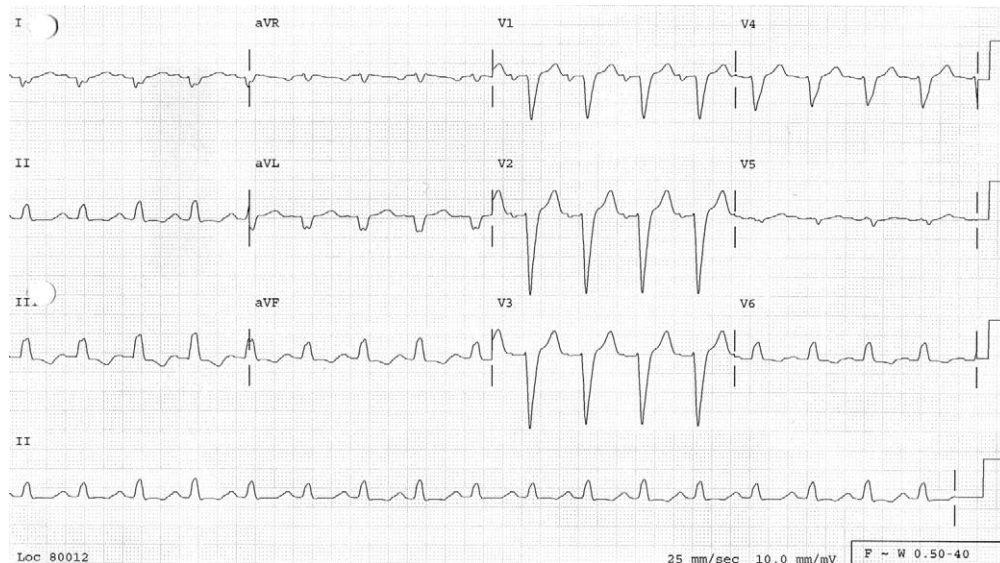


Figure 1. ECG showed sinus tachycardia, right axis deviation, intraventricular conduction delay, poor R-wave progression, long QT interval, and inferior ST-T abnormalities.

edema was observed in both lower limbs. He had grade 5 strength in his upper limbs bilaterally and grade 2 to 3/5 strength in his hip abductors/adductors bilaterally.

Laboratory Work-up

A 12-lead electrocardiogram (ECG) showed sinus tachycardia, right axis deviation, nonspecific intraventricular conduction delay, left atrial enlargement, poor R-wave progression, long QT interval, and inferior ST-T abnormalities, indicating possible ischemia (Figure 1). A chest radiograph showed marked left ventricular enlargement.

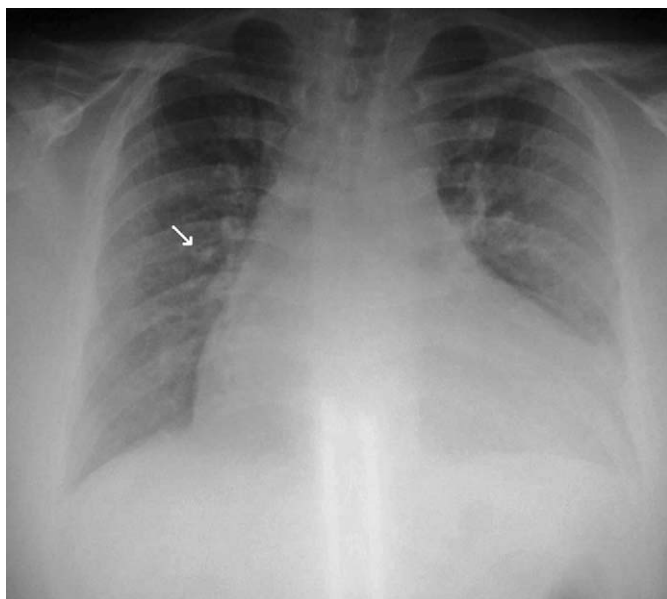


Figure 2. Chest radiograph showed marked left ventricular enlargement. Peribronchial cuffing (arrow) indicated perivascular interstitial edema.

Peribronchial cuffing (arrow) indicated some degree of early perivascular interstitial edema (Figure 2). Transthoracic echocardiogram showed a severely dilated left ventricle with severe global hypokinesia. Left ventricular wall thickness was normal. Left ventricular systolic function was severely reduced (Figure 3). Left ventricular ejection fraction was less than 20%. The right ventricle was moderate to severely dilated, and systolic function was moderately reduced. The left and right atria were moderately dilated. Mild to moderate mitral regurgitation and tricuspid regurgitation were also present.

Overnight pulse oxygen monitoring showed 217 episodes of desaturation with the lowest reading at 76%. The total duration of oxygen saturation less than 90% was 1 hour and 39 minutes. History and subsequent studies were consistent with a diagnosis of chronic

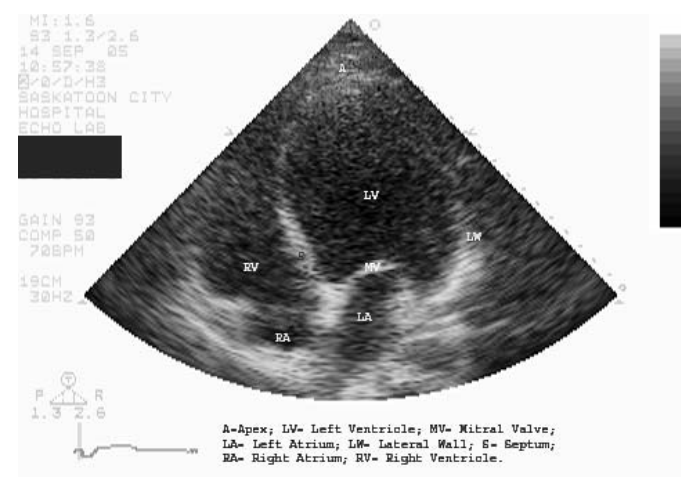


Figure 3. Transthoracic echocardiogram showed severe dilation of the left ventricle with marked global hypokinesia.

obstructive pulmonary disease, obstructive sleep apnea (OSA), and severe cardiomyopathy resulting in congestive heart failure. After furosemide administration, the patient immediately became less dyspneic and more involved in ADL activities. Treatment with warfarin and ramipril was also initiated. Two days later, he was prescribed continuous positive airway pressure (CPAP) for OSA. At discharge, cardiovascular and respiratory symptoms, as well as strength and endurance, had improved, and he was able to manage wheelchair transfers independently.

Approximately 1 year after discharge, his weight had decreased to 94 kg (BMI, 29.7 kg/m²), his ADL functioning and endurance had improved, and his fatigue level had decreased. He was able to propel his wheelchair for short distances without experiencing shortness of breath, and he had been able to return to work. A referral for a sleep study was also made. He remained on anticoagulant therapy. A repeat transthoracic echocardiography showed that the ejection fraction was still low (<20%). Severe left ventricular enlargement with severe global left ventricular systolic dysfunction was noted. Trace mitral regurgitation was present. These findings were compatible with the diagnosis of dilated cardiomyopathy.

DISCUSSION

Cardiomyopathy, especially dilated cardiomyopathy, is recognized as a significant cause of heart failure in the normal population (18). Signs and symptoms of cardiomyopathy, which are similar to those of heart failure, include shortness of breath, progressive dyspnea on exertion, orthopnea, leg edema, paroxysmal nocturnal dyspnea, jugular venous distension, systolic murmurs of mitral and tricuspid valve regurgitation, enlarged and pulsatile liver, S₃ or S₄ gallop, and displaced apical impulse (19). Individuals with dilated cardiomyopathy often present with symptoms of congestive heart failure, systemic or pulmonary emboli, fatigue, and weakness (20). Dilated cardiomyopathy can be subdivided into 9 subcategories based on the cause: ischemic cardiomyopathy, idiopathic dilated cardiomyopathy, toxic cardiomyopathy (alcohol or illicit drugs), hypertensive heart disease, infective cardiomyopathy, valvular heart disease, peripartum cardiomyopathy, tachycardia-induced cardiomyopathy, and cardiomyopathy secondary to metabolic conditions (19).

Typically, individuals with lumbar SCI do not present with cardiovascular compromise. Blocker et al (21) studied the resting ECG of 98 individuals with chronic SCI and reported that individuals with lumbar SCI, unlike those with cervical and thoracic SCI, had no ECG abnormalities. This finding was surprising, considering that patients with chronic SCI are often overweight or obese (22) and certainly at a higher risk for various cardiovascular diseases. The prevalence of overweight (proportion of patients with BMI of 25–29.99 kg/m²) and obesity (proportion of

patients with BMI \geq 30 kg/m²) among individuals with paraplegia and ASIA score C was 70.83% and 50%, respectively. According to the American Heart Association scientific statement, obesity is an independent risk factor for cardiovascular diseases (23). Excessive body weight increases metabolic demand and results in increases in total blood volume, cardiac output, and stroke volume. An increase in left ventricular filling pressure and volume may eventually lead to chamber dilation or hypertrophy.

In addition, sleep-related breathing disorder is a potential risk factor for cardiovascular disease (24–27). Shamsuzzaman et al (28) completed a systematic review regarding the interactions of OSA with cardiovascular pathophysiology and disease. They concluded that repetitive apneic events in patients with OSA may disrupt the normal physiologic interactions between sleep and the cardiovascular system. The activation of a number of neural, humoral, thrombotic, metabolic, and inflammatory disease mechanisms (ie, chronic sympathetic activation, endothelial dysfunction, increased levels of C-reactive protein and inflammatory cytokines, elevated leptin level, hypercoagulability, as well as increased production of endothelin and catecholamines) may induce structural cardiac changes that may be implicated in the initiation and progression of arrhythmia, cardiac ischemia, congestive heart failure, and stroke (28).

Various studies (29–32) have documented a high prevalence of sleep apnea among individuals with tetraplegia. Bach and Wang (29) found that at 5 years after injury, 5 of the 10 individuals with stable traumatic tetraplegia had an increased frequency of transient nocturnal oxyhemoglobin desaturations. Hypercapnia was also more common in this study (29). Burns et al (30) examined the prevalence of sleep apnea among 20 men with various levels and degrees of chronic SCI and found a greater prevalence of sleep apnea with tetraplegia. In a cross-sectional study, Leduc et al (31) reported that 22 of 41 individuals with chronic cervical SCI had OSA-hypopnea syndrome. Berlowitz et al (32) also studied the incidence of sleep disordered breathing among 13 subjects in the first year after cervical SCI and concluded that 62% of the subjects had sleep-disordered breathing within 4 weeks of the injury.

Because of other comorbidities, differentiating the underlying cause of cardiomyopathy was difficult in this case. Heart failure could have been precipitated by a combination of causes because he had many risk factors including smoking, hypertension, poor diet, chronic obstructive pulmonary disease, and long-term obesity (33). All these factors are also well-known risks for cardiovascular diseases in the general population.

OSA likely also contributed to his fatigue. However, the primary causes for his symptoms was felt to be cardiomyopathy and congestive heart failure, because his fatigue levels and shortness of breath improved immediately after administration of furosemide, even before his

use of CPAP. However, in the long term, CPAP also had a positive effect on his symptoms.

CONCLUSION

This report illustrates an obese patient with chronic SCI and severe, progressive fatigue that interfered with his ability to work and perform ADLs. Diagnostic work-up showed severe dilated cardiomyopathy. Treatment of cardiomyopathy, congestive heart failure, and sleep apnea resulted in an improvement of symptoms and in ADL function.

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