Cardiac Cachexia Caused by Right Ventricular Outflow Tract Obstruction in a Patient With Severe Pectus Excavatum

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External compression of the right ventricle (RV) due to a depressed sternum in patients with pectus excavatum is uncommon. Moreover, mid-RV obstruction-induced cachexia rarely occurs in patients with pectus excavatum. We report a case of cardiac cachexia caused by significant RV compression in a patient with pectus excavatum. (Korean J Med 2012;83:637-640)

Keywords: Cachexia; Funnel chest; Ventricular dysfunction, Right

INTRODUCTION

Funnel chest or pectus excavatum (a Latin term meaning hollowed chest) is one of the most common congenital anomalies. It is characterized by posterior depression of the sternum and adjacent costal cartilage. In severe cases, it directly impinges on the right ventricle (RV) [1,2].

Cardiac cachexia is a common complication of chronic heart disease, which is associated with a poor prognosis, independent of functional disease severity, age, measures of exercise capacity, and left ventricular ejection fraction [3].

A case of RV dysfunction that was caused by pectus excavatum has been reported in Korea, but no case report of cardiac cachexia caused by pectus excavatum is known. We report a case of severe mid-RV compression and cardiac cachexia caused by pectus excavatum.

CASE REPORT

A 48-year-old woman presented to the hospital complaining of worsening dyspnea over the last 5 years. Her blood pressure was 100/60 mmHg, pulse rate was 50 beats per
minute, and respiratory rate was 20 beats per minute at first presentation. She was very cachectic and showed a much lower body weight as compared with the average weight for her age, with a height of 157 cm and weight of 28 kg (Fig. 1). Her body mass index was 11.35 kg/m². She was 45 kg in her 20s and had lost about 1 kg per year for the last 10 years. A physical examination revealed that her lower sternum was posteriorly dislocated and concavely deformed, resulting in a funnel-shaped thorax. Auscultation of the chest wall revealed a harsh systolic grade V-VI/VI murmur at the left upper stern- 

![Figure 1](image1.png)

**Figure 1.** Photograph shows the general appearance of the patient.

![Figure 2](image2.png)

**Figure 2.** Transthoracic echocardiographic examination shows a mosaic pattern suggestive of flow acceleration from the mid-cavity of the right ventricle (RV) to the RV outflow tract in the mid-systolic phase on parasternal short axis color Doppler images at the aortic valve level.

![Figure 3](image3.png)

**Figure 3.** Three-dimensional echocardiography shows a not fully relaxed mid-cavity of the right ventricle (RV), even in the diastolic phase. The measured mid-RV dimension was 0.65 cm during diastole.

![Figure 4](image4.png)

**Figure 4.** Contrast-enhanced chest computed tomography scan shows a definitely compressed mid-right ventricular cavity near the sternum. The calculated Haller index (A/B) was 11.25.

Initial laboratory findings were decreased hemoglobin (9.4 mg/dL) and a platelet count of 110,000/mm³ (normal range, 150,000-450,000/mm³) without evidence of coagulopathy (prothrombin time, 13.9 sec; activated partial thromboplastin time, 32.2 sec). Her albumin level was also low (2.9 g/dL). To evaluate the causes for the weight loss and anemia, we performed an esophagogastroduodenoscopy and abdominal computed tomography (CT), which revealed no abnormalities.

An initial electrocardiogram showed sinus bradycardia (50
beats per minute), a left axis deviation with an inferior axis, and low voltage in the limb leads. The patient’s heart was displaced toward the left and posterior side by a concavely dislocated lower sternum on chest posterio-anterior and left lateral view X-rays, respectively.

A transthoracic echocardiographic (TTE) examination showed a normal left ventricular (LV) ejection fraction (64% by the modified Simpson’s method) with normal systolic and diastolic LV dimensions; however, RV hypertrophy was revealed. Color Doppler images of the parasternal short axis view at the aortic valve level showed a mosaic pattern suggestive of flow acceleration from the mid-cavity of the RV (Fig. 2, arrow) to the RV outflow tract in the mid-systolic phase. No significant tricuspid regurgitation was observed. A continuous wave Doppler image showed an intraventricular pressure gradient of 13 mmHg with an intraventricular peak systolic velocity of 1.8 m/s in the RV. The calculated RV stroke volume was 160 mL, and cardiac output was 7 L/min. A three-dimensional TTE examination showed severely limited (0.65 cm in diameter) expansion of the RV mid-cavity during the diastolic phase (Fig. 3, arrow).

We performed a chest CT to evaluate the severity of the pectus excavatum and to assess its anatomical relationship with surrounding organs. The mid-portion of the RV was clearly compressed by the sternum, and the Haller index was 11.25 (Fig. 4).

Severe osteoporosis on bone densitometry was noted. We diagnosed near obstruction of the mid-RV by pectus excavatum and cachectic morphology. We suggested that the generalized loss of lean, fat, and bone tissue was due to uncompensated cardiac performance from chronic disease-induced anemia as a result of pectus excavatum-induced severe mid-RV compression. We recommended an operation for the chest anomaly, and the patient is currently preparing for the operation, including correction of osteoporosis.

**DISCUSSION**

Pectus excavatum is a common congenital anomaly with a prevalence of approximately 1 in 400 births [1]. The pathogenesis of pectus excavatum remains unclear, but the most prevalent theory is that the deformity results from unbalanced overgrowth in the costochondral regions [1,2].

During the adolescent growth spurt, symptoms become increasingly apparent and include easy fatigability and shortness of breath with mild exercise. Electrocardiographic abnormalities are common in these patients, consisting primarily of axis deviation and depressed ST segments, which reflect rotation of the heart within the thorax rather than an intrinsic abnormality [1]. Compression of the RV outflow tract may cause a functional systolic cardiac murmur along the upper left sternal border in approximately 18% of patients [4]. The extent of RV morphological abnormalities is associated with severity of the chest wall deformity. These abnormalities include localized sacculations of the RV wall, a rounded RV apex, trabecular hypertrophy, and/or structural changes of the moderator band. In contrast, aortic and left heart dimensions are similar to those of normal subjects [5]. The severity of pectus excavatum deformities can be calculated by dividing the inner width of the chest at the widest point by the distance between the posterior surface of the sternum and the anterior surface of the spine as determined on CT or chest radiographs. The mean value is about 2.52, and patients with pectus excavatum will have a range of index measurements from 3.2 to 12.7 [5].

The optimal age for repair appears to be 12-16 years; however, adults with persistent pectus excavatum deformities extending into the fourth and fifth decades have achieved excellent results following repair [5].

Cardiac cachexia is a common complication of chronic heart disease [3]. Body composition analyses of body fat and lean tissue estimates, anthropometric measurements (skin fold thickness, arm muscle circumference, and body mass index), serum albumin concentration, and a history of weight loss have all been used to diagnose cachexia. Anker et al. suggested a definition of “clinical cardiac cachexia” as a patient with chronic heart disease of at least 6 months duration without signs of other primary cachetic states. Cardiac cachexia
can be diagnosed when weight loss > 7.5% of the previous normal weight is observed for > 6 months [3]. The patient in this case showed almost normal cardiac output and no evidence of RV pressure overload, such as tricuspid regurgitation, right atrial enlargement, or post-obstructional dilatation. These results were obtained despite a compressed mid-RV. However, considering that she had significant anemia (9.4 mg/dL), cardiac output and/or heart rate should have increased to compensate for the decreased oxygen supply to organs. We suggest that this is the plausible cause of the cardiac cachexia in this patient. This case illustrates that even without measurable heart failure by conventional diagnostic methods, patients with chronic heart disease can suffer cardiac cachexia.

In this case report, we presented a patient who complained of dyspnea and showed cachectic morphology with a harsh mid-systolic murmur on initial presentation and diagnosed pectus excavatum with severe RV compression (Haller index, 11.25). No evidence of other chronic illness was noted. As far as we know, this is the first case report of cardiac cachexia due to severe RV compression during diastole in a patient with a funnel shaped chest.

CONCLUSION

We suggest that even without measurable heart failure by conventional diagnostic methods, patients with chronic heart disease can suffer cardiac cachexia. The plausible mechanism is uncompensated cardiac output despite chronic disease-induced anemia.

REFERENCES