

Acromegaly Cardiomyopathy Of A Young Patient At Initial Stage: A Case Report

M Aparci, E Kardesoglu, N Oezmen, B Cebeci, O Uz, E Demiralp

Citation

M Aparci, E Kardesoglu, N Oezmen, B Cebeci, O Uz, E Demiralp. *Acromegaly Cardiomyopathy Of A Young Patient At Initial Stage: A Case Report*. The Internet Journal of Cardiology. 2005 Volume 3 Number 2.

Abstract

Acromegaly is a chronic endocrine disease caused by growth hormone excess. It can lead to cardiomyopathy, a clinical condition causing increased cardiovascular morbidity and mortality. Patients with acromegaly remained untreated may develop a wide spectrum of cardiovascular abnormalities including myocardial hypertrophy, hypertension, metabolic syndrome, conduction disturbances, dilated cardiomyopathy and congestive heart failure. High suspicion index of cardiac involvement in patients with acromegaly should be maintained. Management of primary disease will improve the patients' prognosis and the quality of life.

In this case we present a young patient with acromegaly at initial stage of acromegaly cardiomyopathy. We aim to remind the importance of cardiomyopathy complicating the acromegaly.

INTRODUCTION

Acromegaly is a rare chronic endocrine disease commonly caused by growth hormone (GH) secreting pituitary adenoma.¹ It is associated with increased morbidity and mortality mainly due to the cardiovascular complications. Wide spectrum of cardiovascular disorders is encountered in acromegaly through abnormal myocardial hypertrophy, diastolic dysfunction, endothelial dysfunction to dilated cardiomyopathy and congestive heart failure in advanced disease.³

CASE REPORT

A twenty one year-old male, complaining of digital overgrowth, prognathism, and diastema, was first diagnosed as acromegaly one year ago. He was 182 cm in height and 94 kg in weight. His blood pressure was 110/70 mmHg and the pulse was 80 beat per minute. Abnormal findings were only coarsening of facial features, prognathism, diastema, distal of upper extremities overgrowth. On the cardiovascular examination, apex was palpable and visible at 5th intercostal space on the midaxillary line. S1 and S2 were normal there was no pathological finding on auscultation. Abdominal examination was normal without any sign of organomegaly.

Growth Hormone and insulin like growth factor-1 levels

were 34 ng/ml (normal range 0-1 ng/ml) and 443 ng/ml (normal range 116-358 ng/ml) compatible with the diagnosis of acromegaly. Dynamic MRI of hypophysis revealed left sided macroadenoma (>12 mm) of pituitary in favor of the acromegaly.

Electrocardiogram revealed sinus rhythm with voltage criteria for left ventricular hypertrophy by Sokolov-Lyon criteria (Fig.1)

Figure 1

Figure 1: ECG of the patient

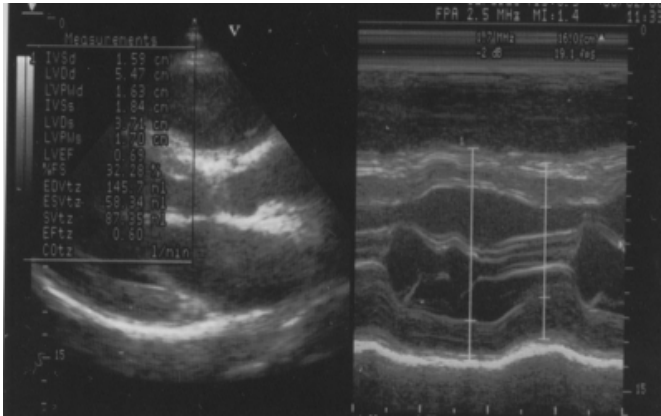


Findings on transthoracic echocardiography were compatible with concentric hypertrophy of left ventricle (LV) without any significant changes in the internal chamber diameters.

Diastolic and systolic thickness of interventricular septum was 15,9 mm and 18,4 mm, respectively and that of left ventricular posterior wall were 16,3 mm and 17 mm, respectively. Diastolic and systolic diameter of left ventricular was 54,7 mm and 37,1 mm, respectively. Left ventricular systolic function was normal with the ejection fraction of 60%. (Figure 2)

Figure 2

Figure 2: Left ventricle, parasternal long axis view showing concentric hypertrophy



Assessment of diastolic function was made by pulse wave Doppler interrogation of the mitral inflow. Mitral valve E and A velocities were 1 m/sec and 0.43 m/sec, respectively, E-deceleration time was 256 ms and E/A ratio was 2.3. There was no valvular pathology.

He underwent surgical treatment of pituitary macroadenoma without any supportive medical treatment. Besides the treatment of acromegaly a close follow up with echocardiography for cardiac complications was planned for the patient.

DISCUSSION

Acromegaly, rare chronic endocrine disease caused by GH excess, can lead to significant cardiovascular morbidity and mortality.¹ Heart is an organ on which Growth Hormone (GH) and insulin like growth factor-1 (IGF-1) exerts their detrimental effects. GH and IGF-1 receptors are expressed in cardiac myocytes and cause hypertrophy of cultured myocytes and delay cardiomyocyte apoptosis. Additionally they affect directly myocardial contractility by increasing intracellular calcium and sensitivity to calcium.^{2, 3, 4}

Excess of GH and IGF-1 induces a specific cardiomyopathy characterized by concentric biventricular hypertrophy.⁴ Cardiac hypertrophy involves both the left and right ventricles in 90% of older patients with long duration of

disease. But recent reports suggest that about 20% of young (less than 30 years old) normotensive acromegalics have cardiac hypertrophy and that structural changes can occur even after short-term exposure to GH excess.⁶ Unlike hypertrophic cardiomyopathy, decrease in ventricular diameters does not characteristically accompany the ventricular hypertrophy in acromegaly cardiomyopathy

Early stage of cardiomyopathy is seen in young patients due to a short duration of disease with hyperkinetic left ventricle and increased cardiac contractility and output. Diastolic functions are normal or mildly abnormal, but commonly lead to decrease of cardiac performance on effort. The intermediate stage is the phase in which most adult were recognized with concentric or eccentric hypertrophy and abnormal diastolic filling pattern either at rest or on effort. The late stage is seen elderly patients with impaired systolic and diastolic functions leading to congestive heart failure.⁴ Decreased diastolic filling and impaired ejection fraction response to exercise have also been confirmed by radionuclide studies.³ Congestive heart failure due to acromegaly heart is easily managed with close clinical follow up and effective of treatment of acromegaly whereas the prognosis is poor in the patients remained untreated for a long time for whom heart transplantation is choice of treatment.²

Metabolic complications are frequently seen in acromegaly due to the counteraction of GH to the effects of insulin on glucose and lipid metabolism. Impaired glucose tolerance due to insulin resistance consequently leading to metabolic syndrome are highly prevalent in acromegalics.¹ Endothelial dysfunction is also the main contributing factor of hypertension, atherosclerosis and obesity in acromegaly.⁶ Angina in patients with acromegaly should be evaluated either noninvasive radionuclide imaging test or coronary angiography because of the risk of premature atherosclerotic disease. It is reported that angina may be caused by distal coronary artery disease without any significant lesion on the coronary artery seen on angiography. Ventricular hypertrophy may also contribute to the myocardial ischemia and angina. Valvular pathologies accompany commonly to the acromegalic heart disease but especially in the late stages. Mitral and aortic regurgitations may be prominent with the dilation of ventricles.

Arrhythmias are additional problems frequently seen in acromegalic patients. The patients commonly present with ventricular ectopic beats, paroxysmal atrial fibrillation,

paroxysmal supraventricular tachycardia, sick sinus syndrome, ventricular tachycardia and bundle branch block.₅ Any variable degree of AV block was not reported.

The management of acromegaly and related complications is targeted against hypersecretion of GH through either removal of adenoma by transsphenoidal surgery or controlling tumor growth by radiotherapy. Medical treatment with somatostatin analogues which was an adjunct therapy to surgery is currently being used as first line therapy with significant suppression of GH and IGF-1.₁ However surgical treatment was decided in our case instead of medical treatment. Normal levels of GH and IGF-1 were achieved with values of 1 ng/ml and 243 ng/ml, postoperatively. The patient will be followed and performed echocardiography, yearly.

Our case illustrates a young patient with short duration of acromegaly due to GH secreting macroadenoma associated with cardiac involvement in the form of biventricular hypertrophy, preserved LV systolic and diastolic function.

CORRESPONDENCE TO

Dr.Mustafa APARCI Gata Haydarpasa Training Hospital
Department Of Cardiology Kadikoy, Istanbul, Turkey Tel:
+90 216 542 20 22 +90 505 394 71 31 e-
mail:maparci@gmail.com maparci@hpa.gata.edu.tr

References

1. Ayuk J, Sheppard MC. Growth hormone and its disorders Postgrad Med J 2006;82:24-30
2. Bihan H, Espinosa C, Socin HV, Salenave S, Young J, Levasseur S, Assayag P, Beckers A, Chanson P. Long-Term Outcome of Patients with Acromegaly and Congestive Heart Failure, J Clin Endocrinol Metab, November 2004, 89(11):5308-5313
3. Jaffrain-Rea ML, Minniti G, Moroni C, Esposito V, Ferretti E, Santoro A, Infusino T, Tamburrano G, Cantore G, Rosario C. Impact of successful transsphenoidal surgery on cardiovascular risk factors in acromegaly. European Journal of Endocrinology (2003) 148 193-201
4. Colao A, Vitale G, Pivonello R, Ciccarelli A, Di Somma C and Lombardi G. The heart: an end-organ of GH action European Journal of Endocrinology, 2004;151, 93-101
5. Colao A. Are patients with acromegaly at risk for dysrhythmias? Clinical Endocrinology 2001 55 305-306.
6. Clayton RN. Cardiovascular Function in Acromegaly, Endocrine Reviews, June 2003, 24(3):272-277

Author Information

Mustafa Aparci, M.D.

Specialist of Cardiology, Department of Cardiology, Gata Haydarpaşa Training Hospital

Ejder Kardesoglu, M.D.

Associate Professor of Cardiology, Department of Cardiology, Gata Haydarpaşa Training Hospital

Namik Oezmen, M.D.

Asistant Professor of Cardiology, Department of Cardiology, Gata Haydarpaşa Training Hospital

Bekir Sitki Cebeci, M.D.

Associate Professor of Cardiology, Department of Cardiology, Gata Haydarpaşa Training Hospital

Omer Uz, M.D.

Department of Cardiology, Gata Haydarpaşa Training Hospital

Ergün Demiralp, M.D.

Professor of Cardiology, Department of Cardiology, Gata Haydarpaşa Training Hospital