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An atypical cause leading to a diagnosis of acromegaly

Ivana Ságová[1, 2], Milan Dragula[2], Marián Mokáň[2], Peter Vaňuga[1]

[1]Department of Endocrinology, National Institute of Endocrinology and Diabetology, Ľubochňa, Slovakia
[2]1st Department of Internal Medicine, Comenius University Jessenius Faculty of Medicine, University Hospital Martin, Martin, Slovakia

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Acromegaly is characterized by a chronic hypersecretion of growth hormone (GH). Cardiovascular diseases are the most common comorbidity in this condition [1]. A specific acromegalic cardiomyopathy characterized by a biventricular hypertrophy is the result of a long-term exposure of excessive amounts of GH and insulin-like growth factor 1 (IGF-1) to the cardiomyocyte [2]. We report a case of a patient with an undiagnosed acromegaly, which was firstly presented with an acute heart failure.

A 39-year-old man with a history of arterial hypertension with exogenous obesity was admitted to the department of internal medicine for an acute heart failure. The patient was admitted to the hospital with orthopnoea (in the patient's history there was exertional dyspnoea lasting 3 months, and an orthopnoea and a horizontal position intolerance was presented last night before the hospitalization) without anginal pain and palpitation. He suffered from excessive sweating and arthralgia lasting 1 year. There was no history of alcohol, smoking, or drug abuse. He had a tall stature [height 193 cm, weight 150 kg, body mass index (BMI) 40.5 kg/m^2] and enlarged acral parts of the body (including enlargement of the hands, feet, facial bone, nose, and tongue, with wider space between his teeth). The physical examination showed a blood pressure of 145/90 mmHg, pulmonary auscultation revealed bilateral crackles to the middle of the lungs, cardiac auscultation was normal, and lower limbs were with pitting oedema. 12-lead ECG revealed sinus rhythm at 66 bpm, without ischaemic changes, and without arrhythmia. A chest X-ray showed enlargement of the cardiac silhouette and accentuation of the bronchovascular markings with a maximum in the perihilar and paracardiac regions on the right side. Blood count, coagulation, biochemistry, and inflammatory parameters were normal. At the beginning of the hospitalization, proper treatment of heart failure was started. Echocardiography showed dilated left ventricle and left atrium, hypertrophy of the interventricular septum (IVS 17 mm) with a diffuse hypokinesis, and an ejection fraction (EF) of 22%. Diastolic function was with a restrictive filling pattern. The right ventricle and atrium were also dilated, and the valves were normal (Fig. 1). Coronary angiography showed normal coronary arteries without a stenosis. 24-hour electrocardiographic (Holter) monitoring result was without arrhythmia. The condition was initially assessed as an acute heart failure in poorly treated arterial hypertension, and a diagnosis of a hypertrophic cardiomyopathy was also considered. During the complex treatment of the heart failure (angiotensin converting enzyme inhibitors, diuretics,

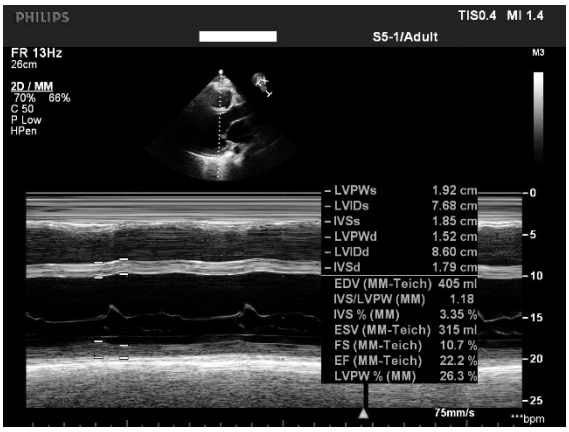

Figure 1. *Echocardiogram on admission. LVPWs — posterior wall thickness end systole; LVIDs — left ventricular internal diameter end systole; IVSs — interventricular septal wall thickness end systole; LVPWd — posterior wall thickness end diastole; LVIDd — left ventricular internal diameter end diastole; IVSd — interventricular septal wall thickness end diastole; EDV — LV end-diastolic volume; ESV — end-systolic volume; FS — fractional shortening; EF — ejection fraction*

Ivana Ságová M.D., Ph.D., Department of Endocrinology, National Institute of Endocrinology and Diabetology, Kollárová 282/3, 034 91 Ľubochňa, Slovakia, tel: +421444306138; e-mail: ivana.sagova1@gmail.com

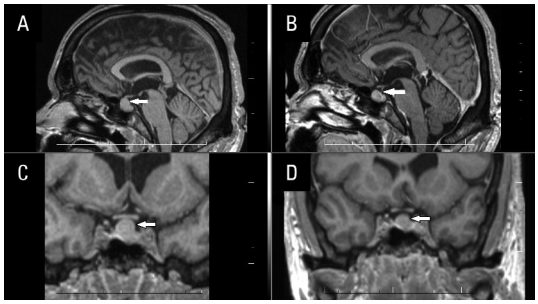

Figure 2. *Pituitary magnetic resonance imaging (MRI).* **A.** *T1VO in sagittal axis — on admission;* **B.** *T1VO in sagittal axis — after 1 year under treatment with somatostatin analogues;* **C.** *T1VO in transversal axis — on admission;* **D.** *T1VO in transversal axis — after 1 year under treatment with a somatostatin analogues. The white arrow marks pituitary adenoma*

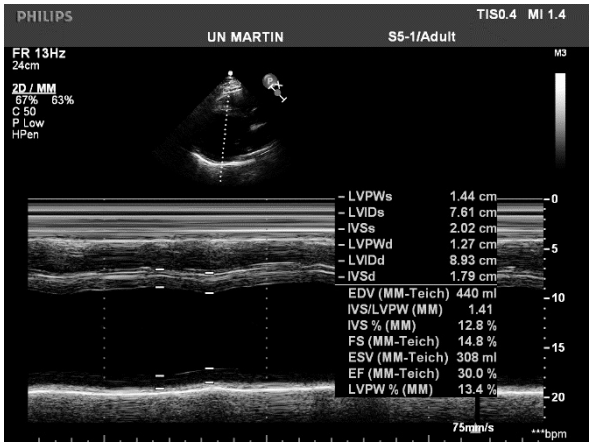

Figure 3. *Echocardiogram after 1 year under treatment with somatostatin analogues. LVPWs — posterior wall thickness end systole; LVIDs — left ventricular internal diameter end systole; IVSs — interventricular septal wall thickness end systole; LVPWd — posterior wall thickness end diastole; LVIDd- left ventricular internal diameter end diastole; IVSd — interventricular septal wall thickness end diastole; EDV — LV end-diastolic volume; ESV — end-systolic volume; FS — fractional shortening; EF — ejection fraction*

β-blockers) the patient's clinical condition improved (NYHA II — during discharge). The patient was subsequently admitted to the National Endocrinology and Diabetology Institute in Ľubochňa for suspected acromegaly. Laboratory tests revealed a high level of GH not suppressed after glucose administration, and high levels of IGF-1 and IGF binding protein 3 (IGFBP3) (Supplementary File — Tab. 1). Magnetic resonance imaging (MRI) scan showed a 14 × 14 × 12 mm macroadenoma of the pituitary gland and enlarged adenohypophysis extending the Turkish saddle. The infundibulum was without deviation, the chiasma opticum was not dilated. The distance of the visual pathway from the pituitary macroadenoma was 0.5 mm. (Fig. 2A, C). A diagnosis of acromegaly was established. A perimetry test ruled out visual field defects. An abdominal ultrasound showed hepatomegaly. Treatment by somatostatin analogues (lanreotide) was initiated at 120 mg every 28 days. Transsphenoidal resection of the pituitary macroadenoma was initially abandoned due to a cardiac finding. After 1 year of treatment with lanreotide the plasma IGF-1 and basal GH normalized. Oral GTT test with GH was with adequate suppression of GH after glucose administration (Tab. 1). A control MRI of the sella turcica demonstrated a regression of the pituitary macroadenoma to size 11 × 11 × 8.5 mm (Fig. 2 B, D). From a cardiological point of view, the clinical condition of the patient improved (functional stage NYHA I). A control echocardiography showed persistent dilatation of ventricles and ventricular hypertrophy, with an improved diastolic function to stage 1 and a slight improvement in systolic function as well as improvement of the cardiac output (Fig. 3). As part of the differential diagnosis of cardiomyopathy, a cardiac MRI was performed, which, due to the patient's size and numerous artifacts, did not further contribute to the diagnosis. From a cardiological point of view, a prospective consultation of the transplant centre is planned in case of insufficient success of the complex treatment.

Cardiovascular disease is a major cause of morbidity and mortality in patients with acromegaly [1]. An early diagnosis and a prompt treatment of acromegaly is essential to avert chronic cardiovascular damage [3]. A strict control of hormone excess in acromegaly is associated with a significant improvement in cardiac hypertrophy, as well as diastolic and systolic functions, leading to an overall improvement in cardiac structure and performance [4]. In the differential diagnosis of a heart failure based on a cardiomyopathy, we must not forget the possible endocrine cause — not only thyroid dysfunction, but also acromegaly.

Conflict of interest

The authors declare that they have no conflicts of interest concerning this article. All authors have read and approved the final form of this article.

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