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Improvement of cardiac function in a Patient with Heart Failure and LVAD placement with treatment of Undiagnosed Acromegaly

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Introduction: This case report explores the improved cardiac function of treating acromegaly in an individual with non-ischemic dilated cardiomyopathy with a left ventricular assist device. The importance of this case study underscores that possible early treatment of acromegalic cardiomyopathy could slow the progression of the cardiomyopathy and prevent invasive cardiac procedures.

Objectives: The primary objective of this review is to underscore a rarely studied effect of on acromegalic cardiomyopathy and subsequently cardiac function.

Methods: This was a retrospective chart review regarding one particular patient case. Epic was used to gather clinical data as well as hospitalization notes/imaging including echo imaging, CT scan. Hormone levels were measured from the time of diagnosis to eventual improvement of cardiac function. Cardiac transthoracic echo was obtained prior to and after starting therapy as part of regular monitoring.

Case Description: 62-year old male with history of non-ischemic cardiomyopathy (EF 15%) with placement of an automated implantable cardiac defibrillator (AICD), pituitary gland tumor, hypothyroidism, and remote history of testosterone deficiency, presented as a transfer to Loma Linda University Medical Center. Prior to his transfer he was admitted multiple times for persistent heart failure exacerbations in Nevada and failed to obtain adequate congestive heart failure (CHF) control.

During the course of his hospitalization in Loma Linda Hospital (LLH), his volume status was optimized and he received right/left heart catheterization with no flow-limiting lesions. It was then decided to proceed with placement of a HeartMate II Left Ventricular Assist device (LVAD). He did well after the placement of the LVAD and continued to undergo further workup for possible future heart transplant. As a part of his ongoing evaluation, Endocrinology was contacted for evaluation due to his history of a pituitary adenoma.

Upon further discussion, the patient stated he was first diagnosed with a pituitary mass in October, 2015. He struck his head on the pavement while getting out of a car. A CT head scan obtained in emergency department (ER) showed a pituitary incidentaloma. The patient denied having visual changes, headaches, weight changes, growth changes, decreased libido, or disruptions in his sleep at that time. Patient stated he was also diagnosed with low

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testosterone about four years prior and was previously on testosterone replacement therapy. He was further diagnosed with hypothyroidism and has been on continued levothyroxine therapy.
He was subsequently seen by an endocrinologist in Nevada for the pituitary mass but no other hormonal deficiency or excess was diagnosed at that time. He then had an MRI of the brain done that showed a 2.3x2.4x2.6cm macroadenoma invading the cavernous sinus with indention of the chiasm and a displaced pituitary stalk.

While undergoing evaluation by LLU Endocrinology, his physical exam was significant for coarse facial features, large hands, mild frontal bossing and some widened spacing of his teeth. This led to suspicion for acromegaly. Further testing included a GH hormone suppression test which confirmed the diagnosis of acromegaly due to a GH secreting pituitary macroadenoma. The patient was started on lanreotide injection. After several months of treatment, his IGF1 levels improved significantly, but did not completely return to normal. He was then started on cabergoline which then did bring GH levels down to normal. On 12/21/17, a repeat TTE showed a significant improvement of his left ventricular ejection fraction, with increase to approximately 40%. This improvement is thought to be, at least in part, due to normalization of his GH levels.

Discussion: Cardiovascular complications are relatively common in patients diagnosed with acromegaly. Studies have reported early development of endothelial dysfunction and structural vascular alterations, with subsequent increased risk of coronary artery disease (Mosca). Based on literature, it is estimated that about 3-10% of acromegalic patients developed acromegalic cardiomyopathy leading to congestive heart failure (Bihan). Acromegalic cardiomyopathy is characterized by concentric biventricular hypertrophy, diastolic dysfunction, and eventually progressive impairment of systolic performance leading to overt heart failure. In these patients, it is believed that chronic exposure to high levels of GH and IGF-1 was the underlying cause, and thus early control of GH and IGF-1 excess has been crucial in management of such patients. The patient we discussed, highlights this point as the improvement of LVEF from 15% to 40% after initiation of Lantreotide and Cabergoline further supports these previous studies.

Despite stabilization of heart failure with control of GH and IGF-a, heart transplantation remains the final solution to end stage acromegalic cardiomyopathy. There has been anecdotal case reporting of heart transplantation in acromegalic patients with heart failure (Albat), and a case reported on acromegalic cardiomyopathy associated malignant arrhythmogenic pattern also successfully treated with heart transplantation (Doimo). However, due to the rarity of the disorder, there has not been any case involving an LVAD in the treatment of acromegalic cardiomyopathy. The use of LVAD in our patient allows time for cardiac optimization while attempting to control GH and IGF-a, as evidenced by an increase in LVEF from 15% to 40%. Such improvement allows more time to modify other risk factors, such as smoking cessation and improve BMI in our patient, therefore increasing patient's chance of survival while waiting for heart transplantation.

Conclusion: Severe heart failure patient secondary to non-ischemic cardiomyopathy with clinical features of acromegaly or

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incidental finding of a pituitary adenoma, need to be evaluated for acromegalic cardiomyopathy. If IGF-a and GH are confirmed to be elevated, it is important to initiate medication therapy for optimal control. In addition, the use of LVAD in patients initially not deemed a candidate for heart transplantation can provide more time for risk factor modification, which may eventually give them a second chance to receive heart transplantation.

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