A MASQUERADE OF ACROMEGALY WITH UNDERLYING HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY

Blair Tilkens, DO, Dajun Wang, MD, Joaquin Solis, MD, John D Crouch, MD, Heather K Sanders, NP, Abdul Jamil Tajik, MD
Advocate Aurora St. Luke's Medical Center, Milwaukee, WI

BACKGROUND

• Acromegaly is a rare endocrine disorder that is commonly caused by a growth hormone (GH) secreting somatotroph pituitary adenoma leading to increased levels of insulin-like growth factor 1 (IGF1) production.
• Increased GH and IGF1 secretion can lead to cardiac hypertrophy.
• We present a case of acromegaly with hypertrophic obstructive cardiomyopathy (HOCM).

CASE

• 61 year old female presented with dyspnea.
• She had coarse facial features with prognathia, macroglossia, and enlarged hands. 4/6 systolic ejection murmur at the left sternal border and 4/6 holosystolic murmur at the apex were noted.
• IGF1 was significantly elevated at 639 (ref range 43-222).
• TTE revealed severe asymmetric septal hypertrophy measuring 43 mm with LVOT obstruction of 40 and 75 mmHg at rest and with Valsalva (Fig 1). There was systolic anterior motion of the anterior mitral valve leaflet, flail P3 mitral leaflet, and severe mitral regurgitation.
• Cardiac MRI confirming severe septal hypertrophy with LV mass index of 193 g/m² and patchy fibrosis throughout the septum and anterior wall (Fig 2).

Figure 1: A) Parasternal long axis view with ultrasound enhancing agent demonstrating severe septal hypertrophy measuring 43 mm. B) Parasternal long axis view demonstrating systolic anterior motion of the mitral leaflet.

Figure 2: A) Reverse septal hypertrophy measuring 42 mm B) Patchy mesocardial delayed enhancement scattered throughout the hypertrophied septum and anterior wall with an estimated total burden of 30%. C) T2 mapping demonstrating no myocardial edema. D) T1 mapping showing normal extracellular volume of 21%.

Figure 3: MRI brain T2 weighted image showing large pituitary macroadenoma.

CONCLUSIONS

• Acromegalic cardiomyopathy is a known complication of excessive GH secretion.
• However, the severity of septal hypertrophy in this patient suggested another underlying disease process occurring simultaneously.
• This case highlights the importance of approaching cases with a broad differential. To our knowledge, the association of these two disease processes has not been described previously in literature.

DISCLOSURES

There are no disclosures to report.