CLINICAL VIGNETTE

A Big Heart for Preventative Primary and Specialty Care

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A 25-year-old obese Black male presents to establish care with a primary care physician. He notes a history of elevated blood pressure readings over the past 2 years (maximum reported values of 174/90 mmHg and 150/90 mmHg) and values greater than 140/80 mmHg since early adolescence, although no formal diagnosis of hypertension or treatment was ever given. He denies symptoms of chest pain, shortness of breath, dyspnea on exertion, dizziness, syncope, palpitations, edema, paroxysmal nocturnal dyspnea or snoring. His review of systems was otherwise negative. He strives for a low-salt, balanced diet, however he admits to eating restaurant food often. He exercises regularly by playing ice hockey and weightlifting. Besides a past medical history of being obese as a child, he denies any history of kidney problems, steroid use, and endocrine abnormalities. He is adopted and thus family history of hypertension or sudden cardiac death remains unknown.

On physical exam, his vital signs are 159/88mmHg, heart rate 62 bpm and BMI 32mg/m2. He has a thick neck without evidence of buffalo hump or striae, his cardio/pulmonary exam is within normal limits, there is no leg swelling, and he is neurologically intact. Initial laboratory work reveals mild dyslipidemia, normal renal function, electrolytes, TSH, urinalysis and CBC. In-office ECG shows marked voltage in the chest wall leads consistent with age-corrected-criteria for left ventricular hypertrophy associated with deep asymmetric T-inversions.

Given the childhood onset of hypertension and abnormal ECG suspicious for hypertrophic cardiomyopathy (HCM), a secondary hypertension work-up and cardiomyopathy evaluation was initiated. Renal ultrasound, renin and aldosterone levels, and sleep study; all of which were within normal limits. He was started on an angiotensin-receptor blocker (ARB) and beta blocker for blood pressure control and an echocardiogram and cardiology consult was ordered.

The echocardiogram revealed normal ejection fraction, moderate septal left ventricular hypertrophy with 1.35cm thickness, normal diastolic function, no evidence of systolic anterior motion of the mitral valve or elevated intracavitary and LVOT gradient. There were no other valvular abnormalities noted, no evidence of coarctation of the aorta, and the right ventricle was normal in size, function, with normal pressures. Given the asymmetric left ventricular hypertrophy, ECG findings, and poor visualization of the apex, a cardiac MRI was ordered to further characterize the degree and extent of hypertrophy.

Cardiac MRI demonstrated lower limit of normal systolic function (LVEF: 52%) with concentric left ventricular hypertrophy extending into the apex and septal wall thickness asymmetrically increased relative to posterior and apical walls. The myofibers had a normal array and there was no evidence of delayed enhancement (subendocardial injury). Systolic anterior motion of the mitral valve (SAM) was once again not seen, and there were no increased LVOT gradients. While hypertrophic cardiomyopathy variants can have concentric or apical predominance, the absence of diastolic dysfunction, SAM, and delayed enhancement suggested that the myofibrils were structurally normal muscle cells devoid of abnormal architecture seen in HCM. The normal cells were felt to be a direct response from either his long-standing history of untreated hypertension (hypertensive heart disease) or his heavy weight-lifting history (athletic heart). Given the implications of the different diagnosis, genetic testing panel was sent for variants of HCM, which returned negative.

To ensure the patient didn't have athletic heart, he was asked to abstain from weightlifting for three months and an echocardiogram was repeated which showed regression of LVH. He was ultimately treated for hypertensive heart disease. Lifestyle counseling encouraged cardiovascular exercises and lightweight-high-repetition strategy for muscular tone. A home-cooked, low-salt, Mediterranean diet consisting of 1800 calories per day was recommended. He was continued on ARB and beta blocker therapy with goal blood pressures reached by two months and maintained again one month later.

Discussion

This case illustrates the importance of preventative care to capture potential congenital or acquired cardiovascular conditions that are amenable to early treatment to prevent serious comorbidity. According to 2017 ACC/AHA guidelines, the diagnosis of hypertension in adults ideally will include home blood pressure monitoring, with an office validated device. The diagnosis is made when the mean value is ≥130/80.[1] In the office, the diagnosis can be made in those with just one blood pressure ≥180/120 or ≥160/100 with evidence of end organ damage. When at-home measurements are not possible, at least 3 office blood pressure readings spaced over weeks to months should be used again with a mean value of ≥130/80. Evaluation of hypertension should include electrolytes (including calcium and BUN/creatinine), CBC, TSH, Lipids, EKG, urinalysis with

consideration for urinary albumin-to-creatinine ratio and echocardiogram.

Secondary hypertension testing should be considered in certain scenarios including young age onset, as in this case. Consideration is made for primary kidney disease, primary aldosteronism and sleep apnea syndrome which was evaluated in our patient using renal ultrasound, renin/aldosterone levels and a sleep study. Less common forms of secondary hypertension include oral contraceptives, pheochromocytoma, Cushing's syndrome, coarctation of the aorta, other endocrine disorders, and chemotherapy agents.[2]

Primary hypertension treatment includes lifestyle modification involving low sodium/high potassium diet, weight loss, DASH diet, exercise, and limited alcohol intake. Pharmacotherapy is recommended with sustained home blood pressure measurements ≥135/85 or average office measurements≥140/90. Treating home or in office blood pressures ≥130/80 is indicated with established cardiovascular disease, type 2 diabetes, CKD, age ≥65, or with an estimated 10-year risk of atherosclerotic cardiovascular disease >10%. According to 2017 ACC/AHA guidelines, first line medical options include thiazides, long acting calcium channel blockers, and ACEi/ARB agents. For Black patients, a thiazide or long-acting dihydropyridine calcium channel blocker should be used.

The medical management of this patient presented an interesting scenario as thiazide diuretics and non-dihydropyridine calcium blockers should be avoided in hypertrophic cardiomyopathy patients with left ventricular outflow tract obstruction, secondary to causing decreased preload or peripheral resistance respectively. Thus, an alternate choice of beta blocker and ARB was made for this patient until this possibility could be properly evaluated.

Evaluation with EKG and subsequent echocardiogram and cardiac MRI in conjunction with cardiology consultation were critical in obtaining the accurate diagnoses and proper treatment course for this patient. This approach could ensure effective long-term health outcomes while avoiding potential iatrogenic or preventable morbidity and mortality.

REFERENCES

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