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Extreme Cardiac Hypertrophy in a Male Cadaver in our Human Anatomy Class - A Case Report

Case Report

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Abbreviations: CDC: Center for Disease Control and Prevention; LVH: Left Ventricular Hypertrophy.

Introduction

Cardiomegaly is a term referring to enlargement of the heart [1] in general, but it is not a specific cause of enlargement. Different processes contribute to the development of cardiac hypertrophy, some are normal physiologic responses and others develop from a pathologic etiology. Cardiomegaly is a frequent cause of sudden cardiac death in adults [2]. According to the American Heart Association, heart disease is the number one killer for all Americans. However, Hispanics and Latinos have higher risks of cardiovascular diseases due to high blood pressure, obesity and diabetes. Among Hispanics, hypertension in particular is a major risk factor for heart disease and stroke [3]. In data collected and published by the Center for Disease Control and Prevention (CDC), in the United States it was shown that between Hispanic subgroups including Central or South American, Cuban, Mexican, and Puerto Rican, the Puerto Rican adults were more likely to have two or more chronic conditions like hypertension, coronary heart disease, stroke, diabetes, failing kidneys and others [4]. Hypertensive heart disease comprises a constellation of abnormalities that include left ventricular hypertrophy (LVH), systolic and diastolic dysfunction, and their clinical manifestations including arrhythmias and symptomatic heart failure [5].

LVH can be defined as an increase in left ventricular mass. It can develop as a physiologic response to an increase in the heart's workload-as in training athletes-or more commonly as a pathophysiologic response. The latter can be due to intrinsic factors (cardiomyopathies) or extrinsic factors such as volume overload and high pressure secondary to valvular disease and hypertension. Increasing the stress on the LV wall, when hypertension induced increases in afterload, stimulate myocyte hypertrophy, collagen formation along with fibroblasts, and remodeling of the myocardium with a disproportionate increase in fibrous tissue [6, 7]. The end result is the development of cardiac hypertrophy along with many other vascular changes in the body.

This is a report of a male cadaver whom was found with an enlarged heart weighing 785 g while undergoing dissection in the Anatomy laboratory at San Juan Bautista School of Medicine, Puerto Rico.

Case Presentation

During a cadaveric dissection of the thoracic cavity using a frontal approach after removing the anterior thoracic cage and entering the pericardium at the mediastinum, the great vessels were cut and the heart removed. The following variations were found on a 78-year-old male cadaver. The subject was approximately 6 feet tall and moderately obese. From his medical records there was a history of previous chronic kidney disease, hypertension.

The heart was unmistakably enlarged and weighed 785 g. as compared to normal, length (from apex to base), and thickness (depth, midportion) were measured with a tape measure. Weight was measured with a weighing machine. The mean length, breadth, thickness of heart in males and females are 11.25, 8.78, 3.97 cm,

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and 10.60, 8.31, and 3.63 cm. The mean weight of heart in males and females was 323 and 276 gms [8]. A coronal cut of the heart was done to take the measurements. The left ventricular wall measured 2.2 cm, the right ventricular wall was 0.7 cm and the interventricular septum was 1.4 cm (Figure 1). The left ventricular chamber measured 1.3 cm with papillary muscle hypertrophy and the right ventricular chamber was 5.3 cm. The left atrium lumen measured 4.2 cm and the right atrium lumen was 6.6 cm. The left atrium wall measurement was 0.8 cm and the right atrium was 0.6 cm.

Discussion

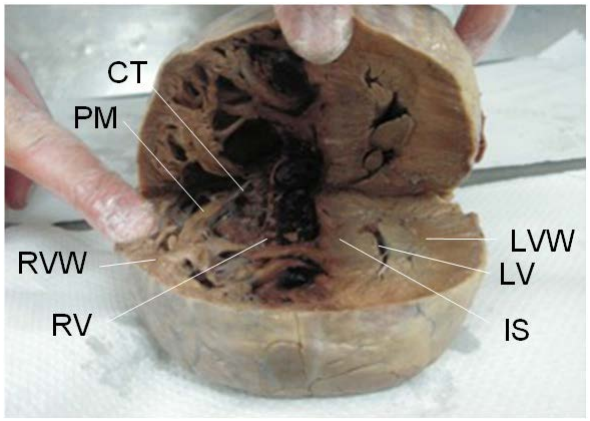
Cardiomegaly is defined as a heart with more than 400 g of weight (Case =785g). Normal heart weight in an adult male ranges from 300 to 380g (Figure 2). On this subject there was notable left ventricular hypertrophy (left ventricle wall = 2.2 cm, normal = 0.6-1.5 cm) [9]. Individuals with LVH present a 2-to-5- fold increase in fatal and non-fatal cardiovascular events when compared with those with hypertension alone [10]. This subject did not present any valvular stenosis or calcification. This is important because of the association with the development of a hypertrophic myocardium. With a myocardium of this size, there is risk of suffering ischemic attacks because of a limited blood supply to the myocardial wall. Arrhythmias are also related to the thickness of the myocardium. Cardiomegaly with presumed hypertensive, multifactorial or unknown cause was the sole arrhythmogenic substrate in 38% of men and 49% of women (p 1⁄4 0.003) [2].

Hypertension and Cardiac Remodeling

Cardiac remodeling, or left ventricle remodeling, are frequently associated with hypertensive subjects [20]. Alterations in size, shape, geometry and function of the heart in response to an injury or load, are the characteristics that define cardiac remodeling [9]. It has been shown that the prevalence of left ventricle hypertrophy (LVH) may vary with the severity of hypertension in each independent patient. It can range from less than 20% in mild hypertensive patients, up to almost 100% in severely hypertensive patients, or patients with complicated hypertension [16]. Historically, left ventricle remodeling has been considered as an adaptive response, to normalize the hemodynamic overload that is imposed on the heart in hypertension [16]. It can be appreciated mathematically by the Laplace Law, $T = P \times r/2h$, where tension on the left ventricle wall (T) is directly proportional to the pressure (P) and radius (r) of the left ventricle, and is inversely directed to the thickness of the wall (h) [11, 20].

Although blood pressure is the most significant factor for left ventricle remodeling, there are many other non-hemodynamic factors that modulate hypertrophic responses in the myocardium, like, ethnicity, gender, neurohumoral, genetic, and environment. [5, 13].

Figure 1. Coronal - sectional cut of heart with hypertrophied left ventricular wall.



Legend: LVW: Left Ventricular Wall; LV: Left Ventricle; RV: Right Ventricle; RVW: Right Ventricular Wall; PM: Papillary Muscle; CT: Chordae Tendinae; IS-Interventricular Septum.

Figure 2. Enlarged heart comparison with age matched normal heart from another cadaver. schizophrenia.

The geometric patterns of the left ventricle can be varied; they are described based on left ventricular mass, which represents hypertrophy, and the thickness of the left ventricular wall relative to the mass [20]. The different classifications of geometric patterns have been identified as: normal geometry of the left ventricle (normal mass and a decrease in the value of relative wall thickness), concentric hypertrophy of the left ventricle (increased mass and relative wall thickness), eccentric hypertrophy of the left ventricle (increased mass and a decrease in the value of relative wall thickness), and concentric remodeling of the left ventricle (normal mass and an increase in the value of relative wall thickness) [14].

The mechanism behind concentric and eccentric hypertrophy is unique for each process. Although the most common pattern detected in a study was normal left ventricular geometry [17], 25% presented with eccentric left ventricular hypertrophy, while a smaller percent presented with concentric left ventricular hypertrophy (18%). Eccentric hypertrophy occurs from increases in the volume of blood, which lead to increases in the radius of the left ventricle [20]. On the other hand, increases in blood pressure lead to an increase in the stress (or tension) the left ventricular wall has to endure in order to push against that high pressure. The problem with exerting tension on the wall is that, myocardial oxygen consumption is dependent on the tension that is being exerted, hence, the higher pressure, higher tension, higher oxygen consumption and demand. This finally leads to a response, in which, the left ventricular wall thickens and increases in mass, in order to stabilize the myocardial stress exerted by the systemic pressure, this structural pattern is known as concentric hypertrophy [20]. These types of evidence, lead us to believe that in hypertensive subjects, the volume overload that presents, is one of the major factors influencing the left ventricular remodeling that occurs [18]. They also have unique characteristics that differentiate each other, not only in the left ventricular geometric patterns, prevalence, and mechanism, but also in histopathological features, renal, and cardiovascular presentations. At the cellular level eccentric hypertrophy is characterized by the addition of sarcomeres in series that result in an increase of the cardiomyocyte cell length. Whereas, an increase in the width of a cardiomyocyte cell is the result of addition of sarcomeres in parallel [12, 15]. There has been some doubt to whether the renin-angiotensin-aldosterone system is one of the major affecting contributors for the remodeling of the left ventricle, but some studies have been able to compare the levels of renin between eccentric and concentric hypertrophy. They demonstrated higher levels of renin associated with concentric hypertrophy, and lower levels of renin with eccentric hypertrophy [5, 13]. At the cardiovascular level, concentric hypertrophy patients have the highest risk of cardiovascular disease; have a lower cardiac output and intravascular volume, which lead to a more of an elliptic shape.

Whereas, eccentric hypertrophy patients have a lower risk of cardiovascular disease, higher cardiac output and stroke volume, lower vascular resistance, and a more spherical shape [13, 20]. In patients that develop heart failure, concentric hypertrophy has been found to be associated with a normal ejection fraction, alternatively, a lower ejection fraction was found on eccentric hypertrophy subjects [21]. There is evidence that suggests that hypertensive subjects may develop dilated heart failure without having prior concentric hypertrophy, which challenges the conventional concept of cardiac remodeling, which establishes that hypertension induces concentric hypertrophy, which eventually is followed by a dilation of the heart chambers, and leads to heart failure [18, 20]. The left ventricular remodeling that occurs as a compensation in response to the overload that is present in hypertensive subjects is directly associated with cardiovascular mortality and morbidity. If the "adaptation" is killing the species, then it really questions to what extent is the left ventricular remodeling acting as a compensatory response or not. But it should not take weight off from the increase it has for cardiovascular events, so left ventricular hypertrophy diagnosis and prevention should be considered in hypertensive subjects [20].

Hypertension and Chronic Kidney Disease

When there is persistent damage in the kidney that is present with a decrease in glomerular filtration rate and accompanied by albuminuria, it is defined as chronic kidney disease. The incidence of chronic kidney disease has increased in a steady manner due to an increase in the number of hypertensive, diabetic, and obesity cases [27]. In 85% to 90% of patients with chronic renal disease (stage 3-5), hypertension has been reported [22]. Hypertension and chronic kidney disease create a symbiotic, cyclic pattern in which one exacerbates the other and vice versa. For example, uncontrolled hypertension is a risk factor for developing chronic kidney disease, developing a faster progression of chronic kidney disease, and additionally, it is the second most common cause of end-stage renal disease [23, 24]. It leads to increased intraglomerular pressure, damaging glomerular filtration. This leads to increases in protein and/or albumin filtration and excretion in the urine, which usually are the first signs of chronic kidney disease. Whereas, progressive renal failure interferes with salt and waste excretion, hence, salt and water retention, leading to volume overload and hypertension. This will increase the blood flow to tissues, which will stimulate autoregulation to occur. Arterioles in the tissues vasoconstrict in response to the increase in blood flow; this will decrease the blood flow, returning to previous flow. This vasoconstriction increases the peripheral vascular resistance, which contributes to the development of systemic hypertension [28].

After explaining the relationship between hypertension and chronic kidney disease, it is important to establish that both are risk factors for the development of each other [27]. Not only risk factors, but they could also be the cause of one or the other. Taking in consideration the case that is presented, in which there is evidence of chronic kidney disease and of hypertension, and the most complicated task is to establish the chronological order of each one.

Either way, the importance is to point out the significance of the association, relationship, or implications hypertension and chronic kidney disease have with each other.

Conclusion

In summary, in the present study we describe a 785 g heart from a 78-year-old male cadaver. The anatomy of the cardiomelic heart is described along with the consequences on the cardiovascular system which are very common among Hispanics and a deeper

understanding of the capabilities of hypertrophy in a heart could help in future studies.

References

[1]. What is an Enlarged Heart (Cardiomegaly)?. Web MD.

[2]. Tavora F, Zhang Y, Zhang M, Li L, Ripple M, Fowler D, et al. Cardiomegaly is a common arrhythmogenic substrate in adult sudden cardiac deaths, and is associated with obesity. Pathology. 2012 Apr;44(3):187-91. doi: 10.1097/PAT.0b013e3283513f54. PubMed PMID: 22406485.

[3]. American Heart Association. Hispanics and heart disease, stroke. 2016.

[4]. Lucas JW, Freeman G, Adams PF. Health of Hispanic Adults: United States, 2010-2014. NCHS Data Brief. 2016 Jun;(251):1-8. PubMed PMID: 27351910.

[5]. Drazner MH. The progression of hypertensive heart disease. Circulation. 2011 Jan 25;123(3):327-34. doi: 10.1161/CIRCULATIONAHA.108.845792. PubMed PMID: 21263005.

[6]. Kahan T, Bergfeldt L. Left ventricular hypertrophy in hypertension: its arrhythmogenic potential. Heart. 2005 Feb;91(2):250-6. PubMed PMID: 15657259.

[7]. Hofmann NP, Giusca S, Klingel K, Nunninger P, Korosoglou G. Severe Left Ventricular Hypertrophy, Small Pericardial Effusion, and Diffuse Late Gadolinium Enhancement by Cardiac Magnetic Resonance Suspecting Cardiac Amyloidosis: Endomyocardial Biopsy Reveals an Unexpected Diagnosis. Case Rep Cardiol. 2016;2016:2461502. doi: 10.1155/2016/2461502. PubMed PMID: 27247807.

[8]. Gupta C, Palimar V, Saxena A, D'souza AS. A morphometric study of measurements of heart in adults and its relation with age and height of the individual: A post-mortem study. CHRISMED J Health Res. 2014 Oct 1;1(4):263-5.

[9]. Dettmeyer RB, Verhoff MA, Schütz HF. Forensic medicine: fundamentals and perspectives. Springer Science & Business Media; 2013 Oct 9.

[10]. Hugar BS, Praveen S, Vinay J, Shetty AR. Sudden death due to coronary artery insufficiency as a result of massive cardiomegaly–a case report. Med Leg J. 2014 Dec;82(4):164-6. doi: 10.1177/0025817213517353. PubMed PMID: 24847045.

[11]. Cohn JN, Ferrari R, Sharpe N. Cardiac remodeling--concepts and clinical implications: a consensus paper from an international forum on cardiac remodeling. J Am Coll Cardiol. 2000 Mar 1;35(3):569-82. PubMed PMID: 10716457.

[12]. Frohlich ED, Susic D. Pressure overload. Heart Fail Clin. 2012 Jan;8(1):21-32. doi: 10.1016/j.hfc.2011.08.005. PubMed PMID: 22108724.

[13]. Dávila DF, Donis JH, Odreman R, Gonzalez M, Landaeta A. Patterns of left ventricular hypertrophy in essential hypertension: should echocardiography guide the pharmacological treatment?. Int J Cardiol. 2008 Feb 29;124(2):134-8. PubMed PMID: 17467083.

[14]. Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. Ann Intern Med. 1991 Mar 1;114(5):345-52. PubMed PMID: 1825164.

[15]. Díez J. Towards a new paradigm about hypertensive heart disease. Med Clin North Am. 2009 May;93(3):637-45. doi: 10.1016/j.mcna.2009.02.002. PubMed PMID: 19427496.

[16]. Ruilope LM, Schmieder RE. Left ventricular hypertrophy and clinical outcomes in hypertensive patients. Am J Hypertens. 2008 May;21(5):500-8. doi: 10.1038/ajh.2008.16. PubMed PMID: 18437140.

[17]. Cuspidi C, Sala C, Negri F, Mancia G, Morganti A. Prevalence of left-ventricular hypertrophy in hypertension: an updated review of echocardiographic studies. J Hum Hypertens. 2012 Jun;26(6):343-9. doi: 10.1038/jhh.2011.104. PubMed PMID: 22113443.

[18]. Osler W. The principles and practice of medicine: designed for the use of practitioners and students of medicine. New York: D. Appleton; 1892.

[19]. Verdecchia P, Angeli F, Achilli P, Castellani C, Broccatelli A, Gattobigio R, et al. Echocardiographic left ventricular hypertrophy in hypertension: marker for future events or mediator of events?. Curr Opin Cardiol. 2007 Jul;22(4):329-34. PubMed PMID: 17556886.

[20]. Nadruz W. Myocardial remodeling in hypertension. J Hum Hypertens. 2015 Jan;29(1):1-6. doi: 10.1038/jhh.2014.36. PubMed PMID: 24804791.

[21]. Velagaleti RS, Gona P, Pencina MJ, Aragam J, Wang TJ, Levy D, et al. Left ventricular hypertrophy patterns and incidence of heart failure with preserved versus reduced ejection fraction. Am J Cardiol. 2014 Jan 1;113(1):117-22. doi: 10.1016/j.amjcard.2013.09.028. PubMed PMID: 24210333.

[22]. Rao MV, Qiu Y, Wang C, Bakris G. Hypertension and CKD: Kidney Early Evaluation Program (KEEP) and National Health and Nutrition Examination Survey (NHANES), 1999-2004. Am J Kidney Dis. 2008 Apr;51(4 Suppl 2):S30-7. doi: 10.1053/j.ajkd.2007.12.012. PubMed PMID: 18359406.

[23]. Botdorf J, Chaudhary K, Whaley-Connell A. Hypertension in cardiovascular and kidney disease. Cardiorenal Med. 2011;1(3):183-192. PubMed PMID: 22096454.

[24]. Segura J, Ruilope LM. Hypertension in moderate-to-severe nondiabetic CKD patients. Adv Chronic Kidney Dis. 2011 Jan;18(1):23-7. doi: 10.1053/j.ackd.2010.11.001. PubMed PMID: 21224027.

[25]. Keane WF, Eknoyan G. Proteinuria, albuminuria, risk, assessment, detection, elimination (PARADE): a position paper of the National Kidney Foundation. Am J Kidney Dis. 1999 May;33(5):1004-10. PubMed PMID: 10213663.

[26]. Yoshioka T, Rennke HG, Salant DJ, Deen WM, Ichikawa I. Role of abnormally high transmural pressure in the permselectivity defect of glomerular capillary wall: a study in early passive Heymann nephritis. Circ Res. 1987 Oct;61(4):531-8. PubMed PMID: 2443267.

[27]. Buffet L, Ricchetti C. Chronic Kidney Disease and Hypertension. Medscape. 2016 Oct 7.

[28]. Salem MM. Pathophysiology of hypertension in renal failure. Semin Nephrol. 2002 Jan;22(1):17-26. PubMed PMID: 11785065.