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Obesity Related Cardiovascular Diseases and Diagnosis

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Review Article

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ABSTRACT

Obesity is defined as a condition in which the amount of adipose tissue in the body is excessive and there has been many researches proving a strong correlation between cardiovascular diseases in obese individuals. The elevated incidence of many identified risk factors including hypertension (HTN) ,diabetes mellitus (DM), and hyperlipidaemia, it could be single and as disease is transmitted to a degree that is indirect great degree for higher incidence of cardiovascular disease in obese people. Obesity, on the other hand, has numerous direct effects on the cardiovascular system, which will be explored in depth. We also look at how to overcome the numerous problems that obesity poses in the performance and interpretation of cardiac tests. Adiposity raises danger of unfavourable cardiovascular system (CVS) problems in a variety of ways. These effects may be mediated indirectly by metabolic syndrome danger elements such as hyperlipidemia, HTN, and dysglycemia, or by the impact of adiposity-related sleep disturbances. Dysmetabolic syndrome are linked to truncal adiposity, associated fat spread mostly part of the abdomen as compared to periphery. The circumference of waist are effective measures of adipose spread and higher amount

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indicate a higher danger of cardiovascular disease. However, there are several direct consequences of adiposity on CVS that aren't influenced by metabolic syndrome factors or related effects of sleep disturbances. This essay will mostly focus on these topics. Because of restrictions in physical examination as well as different tests such as electrocardiograms (EKGs), imaging examinations, and cardiac catheterization, obesity also makes it difficult to make an accurate cardiovascular diagnosis. We'll talk about these restrictions and offer suggestions for how to overcome them.

Keywords: Obesity; congestive cardiac failure; cardiac arrythmia; hypertension.

1. INTRODUCTION

Obesity is defined as a condition in which the amount of adipose tissue in the body is excessive and there has been many researches proving a strong correlation between cardiovascular diseases in obese individuals. The elevated incidence of many identified risk factors including HTN, DM, and hyperlipidemia, it could be single and as disease is transmitted to a degree that is indirect great degree for higher incidence of cardiovascular disease in obese people. Obesity, on the other hand, has numerous direct effects on the cardiovascular system, which will be explored in depth [1-2].

Incidence amount has raised of different acute phase reactant in abdominal obesity, also in emergency condition like hypercoagulability. In obese patients, many adipokines and other chemical mediators such as tumor NF-alpha, IL-6, plasminogen activator inhibitor-1, resistin, lipoprotein lipase, acylation stimulating protein, cholesteryl ester transport protein, retinal binding protein, estrogens, leptin, angiotensinogen, and insulin-like growth factor-1 are present in higher concentrations [2-3]. These have a variety of negative impact in CVS, including producing arterial wall damage and more blood pressure in vessels, as well as promoting a pro-inflammatory and prothrombotic condition.

2. PATHOPHYSIOLOGY OF CARDIO-VASCULAR SYSTEM IN OBESE PEOPLE

The fat mass's having perfusion about 2-3 mL/100 g/MIN, although it rise by about ten times following meal consumption [1]. Perfusion per unit mass, on the other hand, diminishes when obesity grows. When the amount of fat in the body weight grows from 20% to 36%, the cardiac output drops from 2.36 mL/min to 1.53 mL/min, indicating that The increase in c. output isn't proportionate to the overall amount of adipocytes in the body [2]. An increase in stroke volume is used to boost c. output in person with abnormal BMI in order to fulfil the metabolic needs of the adipose tissue. There eccentric form of hypertrophy of the LV, which increase in size to meet the elevated blood returning to heart, to keep the wall tension in normal limit [3]. The left atrium also grows in size in obese people, first owing to increased venous return and blood volume, but subsequently due to abnormal diastolic function and LV dilatation [4]. However, as the Strong Heart Study cohort, increase in isovolumic output and LV hypertrophy in fat person is shown to be associated with accompanying rise in Compared to the quantity of adipose on the body, lean body mass is more important [5].

The left ventricular filling pressure rises with activity, often to more than 20 mm Hg, even if it is normal at rest. The eccentric form of hypertrophy occurs in the left ventricle, although it is less prevalent [6]. Obesity-related cardiomyopathy is because of direct effect cause of adiposity on CVS. Rise in cardiac adipocytes content is first due to a neoplastic change. Fat cells replace many cardiac components, including a SA node, AV node, RBB, and cardiac muscle near AV ring. Conduction abnormalities such as atrisinal block, BB block, and in rare cases, AV block can occur as a result of these [7]. As a result, uneven bands of adipose tissue may split, causing myocardial cell atrophy due to strain. Fat cells can produce regional functional chemicals like adipokines, that can harm neighbouring cardiac cells indirectly. Triglyceride accumulation in normal cells such as muscle cells induce cell function abnormality due to lipid toxicity [8].

2.1 Congestive Cardiac Failure and Obesity

Obesity alone causes 11 percent of cardiac insufficiency among males and 14 percent of

cardiac insufficiency in females in the community [9].

According to the scientific community, obesity is a questionable risk factor for cardiovascular disease. Obesity has been related to cardiac structural and functional problems, as well as deleterious effects on hemodynamic and the structure and function of the left ventricle (LV). It is plausible to assume that obesity contributes to an increase in the incidence and prevalence of heart failure [10].

Obesity causes an increase in total amount of blood, c. output, but it causes a decrease in systemic aversion to flow of blood. These are thought to be adaptive processes that help to keep the body in a state of homeostasis. The increase in circulating amount of blood lead to elevated stroke volume &, as a result, more c. output. Although sympathetic stimulation may occasionally cause a modest increase HR. the [11-12]. HR typically does not alter considerably.

According to the F. Heart Study almost a 5% elevated in males and a 7% increase in women's chance of getting HF per unit of BMI [13]. The length of morbid obesity had an effect on the prevalence of heart failure, with prevalence rates of 70% and 90% after 20 and 30 years, respectively [14].

Obesity-related metabolic and hemodynamic alterations may influence the occurrence of HF in obese patients [15].

2.2 Mechanism of Heart Failure in Obesity

Hemodynamic changes-more fat and fat less part increase metabolic demand, resulting in high blood flow, increased c. output and increased blood amount in obese people. The back flow to the left & RV increases as blood volume increases, leads to wall pressure & hypertrophy of chambers [16].

Although HR may stay constant / moderately raised, volume elevation in proportionate to extra body mass, resulting to increase in cardiac force more than expected optimal body mass. Because increasing LV pressure and volume more O2 use [17,18] and produce a shift to left in the F-Starling curve [19] the arteriovenous oxygen difference widens. Due to these changes there is a hemodynamic overload which eventually causes left ventricle failure.

3. ALTERATION IN CARDIAC STRUCTURE

3.1 The Association between Heart Mass and Body Mass is Line

LV hypertrophy and dilatation are linked in long-term obesity and systemic hypertension. With the seriousness and period of adiposity, the degree of cardiac remodelling rises [20]. Lauer et al found a substantial positive association between LV mass and BMI, while Rasooly et al found an strong positive connection between LVM and waist circumference [21]. In obesity, there is a proportionate rise in the prevalence of cardiac fibrosis, which is generally accompanied by tissue degradation and inflammation [22]. In the development of LVH and heart dysfunction, myocardial fibrosis is an essential structural change. The vital outcome of adipose produced hormones like leptin [23,24] or the training impact on heart due to excessive body mass that has to be raised throughout regular activities also contribute to development LVH in obese people [23].

3.2 Endothelial Dysfunction and Vascular Changes

Obesity causes atherogenesis as it creates a prothrombotic and proinflammatory state [25]. It's a non-dependent danger for atherosclerosis. Obesity in young person causes atherosclerosis progression and is manifested decades after its onset [25,26,27].

3.3 Cardiac Arrythmias and Obesity

Overweight people had a increase risk of sudden cardiac death &arrythmias. The longer corrected QT (QTc) interval seen in 30 percent of obese patients having glucose intolerance in the NHANES III research. According to Schouten et al. [26] 8 percent of overweight people had a QT interval of greater than 0.44 sec. and 2percent have a QTc interval of greater than 0.46 sec [28].

In obese people there is also an increase in the catecholamines levels Furthermore, higher amounts of FFA in obese people can impair repolarization. It has a link between ventricles having abnormal rhythms and long-chain SFA levels in individuals with myocardial infarction. With weight increase, the autonomic system undergoes a number of alterations. A 10 percent elevation in body mass leads to parasympathetic

attitude to drop & HR to rise. Weight loss, on the other hand, causes a drop-in heart rate. With a ten percent weight decrease, heart rate variability improves significantly. Elevated resting HR & reduced HR irregularity, regardless of ejection fraction, are both predictors of death [29].

In a study of fat persons without clinical cardiac illness, the incidence of late potential (high-frequency, low-amplitude signals at the terminal segment of the QRS complex revealed using high-resolution signal averaged recording) rose proportionately with BMI. Late potentials have been related to an increased risk of ventricular arrhythmias in a range of heart disorders, yet they are observed in only around 3% of healthy persons.. Late potentials were found in 35% of individuals with a BMI of 31 to 40, 86% of those with a BMI of 41 to 50, and 100% of those with a BMI of >50, respectively. This increasing occurrence might be linked to obesity and mononuclear infiltration, as well as fibromyalgia [30].

4. HYPERTENSION

Hypertension affects 15% of males with a BMI of less than 25, and 42% of men with a BMI of more than 30, while it affects 15% and 38% of women, respectively [31]. The multiple of c. output & systemic vascular resistance is blood pressure & in obese people, c. output is enhanced due to more blood supply to the adipose tissue [32]. Because the greater fragmentary region of the arterial floor, we should predict low systemic vascular resistance in overweight people. However, frequently abnormally within normal limit/more, increasing a risk of HTN.33 Low-grade inflammation mediated by adipokines, hyperinsulinemia, and insulin resistance, sympathetic nervous system overactivity, and a disrupted sleep pattern are all variables that promote systemic vascular resistance in obese people. [33] Hypertension becomes more common as fat becomes more severe. It may be diurnal at first, especially if sleep apnoea is present [33].

Filling pressures, systolic pressure, and pulmonary vascular resistance are all higher on the right side. Increased pulmonary vascular resistance can be caused by a number of factors, including intrinsic pulmonary illness, sleep apnoea/hypoventilation, recurrent pulmonary thromboembolism, and left ventricular dysfunction, all of which are more common in obese people [34]. More over half of obese people have raised pulmonary artery pressure, but only to a minor degree [19].

Pertaining to obstructive sleep apnoea, pulmonary hypertension affects 15% to 20% of patients. In the absence of daytime hypoxia, this is usually mild and ranges from 30 to 35 mm Hg. Right ventricular overload manifests very late in the EKG. Obstructive sleep apnoea patients are more likely to experience nocturnal dysrhythmias, right and left heart failure, myocardial infarction, stroke, and death [35].

5. CORONARY ARTERY DISEASE

According to the F-heart research [36] the Manitoba research [37] and the Harvard public health nurse study [38] overweight is an non-dependent indicator of CAD. Patients in the F-cohort ranged in age from 28 to 62 yrs old and are follow up to an average of 26 yrs. When comparing person under the age of 50, a massive class had two times danger of CVS as the leanest class. later accounting the effects of other main CVS more factors, the danger is more2.4 times among overweight women of same age group [36]. Plaques and ulceration in the coronary arteries and abdominal aorta were associated to the amount of abdominal fat and BMI in 15 to 34-year-olds who died from accidental causes [39].

Overweight hasten arterial deposition decennary prior to signs occur & this was true when other danger elements such as more cholesterol, HTN, tobacco abuse & elevated HbA1c were taken into account [36]. Visceral obesity was linked to the solidity of macrophages / milli meter2 of plaques [39]. Obese patients can have higher negative results following coronary artery bypass operation [40]. They had higher risk of postoperative thrombosis, sternum infections, and saphenous vein harvest site infections. A greater rate of atrial arrhythmias is also observed [40]. However, not such a major change in mortality or postoperative cerebrovascular events. Excluding the highly overweight (BMI >35) and when accompanied by diabetes, kidney disease, / age more than 60, pulmonary complications were comparable [41].

6. DIAGNOSIS

6.1 Echocardiogram

Pericardial effusion might be mistaken for a large build-up of subepicardial fat (pseudo pericardial

effusion). Lipomatous hypertrophy in the interatrial septum induced by fat deposition can lead it to thicken to more than 20 mm and even suggest a tumour [42]. Diastolic dysfunction of the left ventricle is fairly prevalent. Subclinical changes in the structure and function of the left ventricle, such as differences in regional or global strain, were discovered in asymptomatic obese individuals several years before they developed signs and symptoms of heart failure as compared to healthy person [43]. In obese people, poor images are frequent, and procedures same as tissue Doppler and pulmonary venous Doppler can help [44].

6.2 Electrocardiogram

The EKG in obese people is influenced by a range of factors including parallel movement of heart by the higher diaphragm, heart enlargement, increased space among the heart and the electrodes, and concurrent sleep disorder/overweight/hypoventilation disease. Voltage drops, towards left axis, horizontal inferolateral T wave, LA hypertrophy, increase false +ve standard for inferior wall MI, & far fewer predominance of LV dilatation may be showed in the EKG than on the basis of echo criterion (only about two thirds [45].

With steep S wave in v3 & towering R wave in a VL, the LV force is much extra posterior and lateral orientated. When compared to echo in detecting left ventricular hypertrophy, a addition of R wave in aVL and S wave in v3, if greater than 35 milli meter in males and 25 milli meter in females, has a sensitivity of 49% & specificity of 93% & much helpful much often preowned voltage criteria [46]. A QRS amplitude may rise, decrease, or remain unchanged as a result of weight loss [47]

6.3 Stress Test and Cardiac Catheterization

In obese individuals, diagnosing coronary artery disease might be tough. Due to resting EKG anomalies induced by overweight and tough doing appropriate activity, stress EKG is problematic. Attenuation artefacts and a higher rate of false positives afflict imaging procedures. Due to the obvious load constraints of the table, cardiac catheterization and radionucleotide imaging are frequently not viable; nevertheless, trans-esophageal dobutamine stress echo may be an excellent, other options. The radial techniques are preferred for cardiac catheterization. The larger amount of fat to be passed through needle to enter the femoral artery via femoral approach [48-51].

7. CONCLUSION

Obesity and overweight represent significant predictors for CVD, especially HF & CHD. Although the precise circumstances linking obesity and indeed the onset of such diseases are unknown, the skill of adipocytes to enlarge and generate pro-inflammatory cytokines which could really straight impede cardiac systolic and diastolic feature and also the establishment of atherosclerotic plaques plays an very important role. Furthermore, additional obesity-related alterations in body composition might cause early cardiac circulation and abnormal development.

Whenever adiposity and HF but rather CHD combine, nevertheless, people who are obese appear to have a good effect than someone who is healthy size or malnourished, notably once CRF is decreased. It's worth noting that perhaps the existence of an obesity dilemma should not have been interpreted as an encouragement of obesity in the community at large or even in people who haven't got CVD. In reality, if overeating could be averted, they might not have gotten that particular CVD during first time, leading to a longer and healthy carefree life of CVD.

A significant cause of elevated CRF is the higher quantity of LM observed in people with the usual obese phenotypes. Increased CRF is linked to a higher chance of survival, which might explain some of the benefits of obese. People with lower levels of LM (sarcopenia) have a higher CRF, even when they are obese (sarcopenic obesity). These findings imply that methods that promote LM, also including cardiorespiratory fitness and changes in food , might be beneficial clinical options. To apply such treatments in medical practices and interest physicians in enabling and recommending clients for such therapies, long-term research assessing the benefits of these therapies on treatment outcome are obviously necessary.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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