Obesity, Obstructive Sleep Apnoea & Cardiac Involvement

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Obesity is a chronic disease that is causally related to serious medical illnesses, impaired quality of life, and considerable economic burden due to increased health care costs and loss of productivity. The global epidemic of overweight and obesity -"globesity" - is rapidly becoming a major public health problem in many parts of the world. It accounts for heavy expenditure in provision of health care[1]. In many developing countries, the progression of nutritional transition has been detected, characterized by a reduction of prevalence of nutritional deficiencies and more occurrence of overweight and obesity.

The typical time sequence of emergence of chronic diseases following the increase prevalence of obesity is important in public health planning. The first adverse effect of obesity to occur in population in transition is hypertension, dyslipidemia and glucose intolerance, while coronary heart disease and the long term complications of diabetes, such as renal failure begin to emerge several years later.

Obesity and particularly central obesity are also potential risk factors for sleep apnea. Obstructive sleep apnea (OSA) is characterized by periodic reduction or cessation of breathing due to narrowing of the upper airways. Obesity can increase pharyngeal collapsibility through mechanical effects on pharyngeal soft tissues and lung volumes, and through central nervous system acting signaling proteins (adipokines) that may affect airway neuromuscular control. Although weight loss reduces upper airway collapsibility during sleep, it is not known whether its effects are mediated primarily by improvement in upper airway mechanical properties or neuromuscular control.

Repeated episodes of hypoxemia, hypercapnia, and microarousal plus intrathoracic pressure fluctuations in Obstructive sleep apnea (OSA) trigger mechanisms such as sympathetic hyperactivity, oxidative stress, systemic inflammation, hypercoagulability, and endothelial dysfunction. OSA episodes produce surges in systolic and diastolic pressure that keep mean blood pressure levels elevated at night. In many patients, blood pressure remains elevated during the daytime, when breathing is normal causing secondary hypertension. These patients have excessive daytime sleepiness, difficulty concentrating on tasks such as driving and remaining focused, generalised irritability & impaired emotional functioning. Other complications that can follow with OSA include heart failure, acute myocardial infarction, arrhythmias, pulmonary hypertension, stroke, and advanced atrioventricular block[2,3].

Cardiac dysfunction can manifest as both systolic as well as diastolic dysfunction, and routinely ejection fraction and conventional Doppler parameters are used for its evaluation. Ejection fraction is a less sensitive parameter in early systolic dysfunction and cannot be used routinely in asymptomatic patients. The sensitivity for the detection of diastolic dysfunction by conventional Doppler parameters is decreased by pseudonormalization pattern in Grade II diastolic dysfunction. This calls for the use of Tissue Doppler-derived indices, i.e., E/E^{0} for differentiation. Myocardial Performance Index (MPI / Tei Index), which includes both systolic and diastolic time intervals to assess the global cardiac dysfunction was used by Tei and his co-workers in 1995. Tei Index uses the measurement possible on flow wave Doppler and is as sensitive as the tissue Doppler measurements. The MPI / Tie index is mainly used in amyloidosis, dilated cardiomyopathy, ischemic heart disease, and congestive heart failure; it is defined as the sum of isovolumic contraction time and isovolumic relaxation time (IVRT) divided by the ejection time. Thus, MPI reflects global LV function as opposed to other measurements that reflect mainly either LV

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systolic or diastolic function. The MPI is a sensitive index of asymptomatic heart failure and predicts future development of heart failure independent of other echocardiographic measurements[4]. Thus MPI can be used as a robust marker of disease severity in patients with OSA and as marker of response to therapy.[5,6]

In this issue of the journal as evaluated by Dr. Yogendra Bansod *et al* myocardial performance index was found to correlate with the severity of obstructive sleep apnoea. Treatment of OSA with nasal continuous positive airway pressure (CPAP) abolishes apneas, thereby preventing intermittent arterial pressure surges and restoring the nocturnal "dipping" pattern. CPAP treatment also has modest beneficial effects on daytime blood pressure. Because even small decreases in arterial pressure can contribute to reducing cardiovascular risk, screening for OSA and MPI can form an essential element of evaluating patients with Obesity.

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