'Angry Fat' and Metabolic Syndrome

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The metabolic syndrome consists of a constellation of abnormalities that confer risk of cardiovascular disease and diabetes mellitus. The syndrome is quite common with an increasing trend worldwide and can be found in approximately one-third of patients with essential hypertension.¹ The rising trend is predominant in the South East Asian Region especially in India and Bangladesh²,³,⁴,⁵ and has been found to be associated with increased atherosclerotic morbidity and mortality. The increasing prevalence of obesity is the driving force behind rising prevalence of the metabolic syndrome.⁶

The major features of metabolic syndrome include central obesity, elevated triglycerides (TG), reduced high density cholesterol (HDLc), high blood pressure and elevated fasting blood glucose.⁷ A recent study conducted in urban community of Bangladesh⁸ revealed prevalence of obesity (BMI≥25) to be about 21% and glucose intolerance 22%. But elevated triglyceride (TG >150mg/dl) was 45% and low HDL-c (HDL <40mg/dl) 43.8%. The crude prevalence of metabolic syndrome was 8.7% according to International Diabetes Federation (IDF) criteria.⁹ However, the prevalence of metabolic syndrome in rural Bangladesh was found to be low (<3%).¹⁰

Components of metabolic syndrome are believed to erupt from a common soil and are threaded together by the Renin–Angiotensin–Aldosterone–System (RAAS). With the rise of body mass index (BMI) blood pressure (BP) increases.¹¹ The risk of new onset of T2DM is doubled in patients with uncontrolled BP and up to 40% of subjects with hypertension have obesity as well.¹² Approximately 70% patients with T2DM also have hypertension and often show evidence of insulin resistance.¹³

Fat, especially visceral fat is a highly dynamic endocrine organ and produces at least three dozens of substances (adipokines) including angiotensinogen. Central fat is also a highest storage organ for Angiotensinogen which is a precursor of a powerful vasoconstrictive agent Angiotensin–II. Fat cells are resistant to insulin’s antilipolytic effect, leading to increased Free Fatty Acid (FFA), which is lipotoxic.¹⁴,¹⁵ With ingestion of more food, central obesity increases and adipocyte’s storage capacity to store fat is also exceeded, and, at time, it becomes dysfunctional.¹⁶ Dysfunctional fat cells then lead to excessive insulin resistance, a component of metabolic syndrome. When dysfunctional fat cells rupture there is “lipid overflow” into the circulation. Atherosclerotic provoking–adipokines that is released by this “lipid overflow” induces inflammatory reaction in different tissues including liver, muscle, pancreas and especially arterial vascular smooth cells, leading to acceleration of atherosclerosis.¹⁷ The term “Angry Fat” is used in such a situation when the adipocytes “burst” leading to excessive unregulated lipolysis causing increased amount of FFA in blood. This leads to fat deposition in various tissues including pancreas, heart and vascular tissue causing injury leading to a cascade of metabolic events like hypertension, T2DM and atherosclerosis.¹⁸

Management of diabetes and hypertension is, therefore, not just the control of blood pressure and blood glucose by pharmacotherapy but should focus on this total metabolic cascade. Pharmacotherapy should include RAAS blockade and proper attention given to other components of metabolic syndrome specially dyslipidemia and obesity for an effective control of total cardiovascular risk.¹⁹,²⁰

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References


