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EDITORIAL

'Angry Fat' and Metabolic Syndrome

M Maksumul Haq[1], ZA Latif[2]

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 Bangladesh[2,3,4,5] 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 Bangladesh[8] 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]

Authors' Information:

[1]***Professor M Maksumul Haq,*** *MBBS, FCPS, FRCP, FACC, Head,Department of Cardiology, Ibrahim Cardiac Hospital & Research Institute, Dhaka.*

[2]***Professor ZA Latif,*** *MBBS, FCPS, Department to Endocrinology & Diabetology, BIRDEM, Dhaka.*

References

1. Redon J, Sifkova R, Laurent S, Nilsson P, Narkiewitcz K, Manica G. The metabolic syndrome in hypertension: European society of hypertension position statement. J Hypertens 2008; 26(10):1891-900.
2. James WP. The epidemiology of obesity: the size of problem. J Inter Med 2008; 263(4): 336-52.
3. Sayeed MA, Mahtab H, Khanam PA, Latif ZA, Banu A, Khan AK. Prevalence of diabetes and impaired fasting glucose in urban population of Bangladesh. Bangladesh Med Res Counc Bull 2007; 33(1): 1-12.
4. Mathen CD. Loncar D. Projection of global mortality and burden of disease from 2002 to 2030. PlosMed 2006; 3(11): 442.
5. Murry CJ, Lopez AD. Mortality and cause for eight region of the world: global burden of disease study. Lancet 1997; 349(9061): 1269-76.
6. Park HS, Kim SM, Lee JS, Lee J & Han JH. Prevalence and trends of metabolic syndrome in Korea: Korean National Health and Nutrition Survey 1998-2001. Diabetes Obes Metab 2007;9:50-8.
7. Oguz A, Sagun G, Ozunlulu M, Alpaslan B, Yorulmaz E, Tekiner E et al. Frequency of abdominal obesity and metabolic syndrome in healthcare workers and their awareness levels about these entities. Arch Turk Soc Cardiol 2008; 36:302-9.
8. Sayeed S, banu A, Khanam PA, Alauddin S, Mokbul S, Begum T, Mahtab H, Sayeed MA. Prevalence of metabolic syndrome in three urban communities of Dhaka City. Ibrahim Med Coll J 2008; 2(2): 44-48.
9. Soto Gonzallz A. Bellido Guerrero D, et al. Does the prevalence of metabolic syndrome improve by applying the International Diabetes Federation Criteria 1. Public Health Nutr 2007; 10(10): 1173-80.
10. Zaman MM, Ahmed J, Choudhury SR, Numan SM, Islam MS, Parvin K. Prevalence of metabolic syndrome in Rural Bangladeshi women. Diabetes Care 2006; 29: 1456-57.
11. Blaj S, Stanciu S, Jurcut C, et al. Hypertension in obese patients: a dysmetabolic hypertension with a possible adipocyte dysfunction mechanism. Rom J Intern Med. 2003;41:103-111.
12. Izzo R, de Simone G, Chinali M, et al. Insufficient control of blood pressure and incident diabetes. Diabetes Care. 2009; 32(5):845-850.
13. Amanda N. Long, DO; Samuel Dagogo-Jack, Comorbidities of Diabetes and Hypertension: Mechanisms and Approach to Target Organ Protection J Clin Hypertens (Greenwich). 2011;13: 244-251.
14. Bays HE, Gonzalez-Campoy JM, Bray GA, Kitabchi AE, Bergman DA, Schorr AB, Rodbard HW, et al. Pathogenic potential of adipose tissue and metabolic consequences of adipocyte hypertrophy and increased visceral adiposity. Expert Rev Cardio Ther 2008; 6: 343-68.
15. DeFronzo RA, Dysfunctional fat cells, lipotoxicity, and type 2 diabetes. Int J Clin Preact Suppl 2004;143:9-21.
16. Salans LB, Bray GA, Cushman SW, Danforth E Jr, Glennon JA, Horton ES, Sims EA. Glucose metabolism and the response to insulin by human adipose tissue in spontaneous and experimental obesity: effects of dietary composition and adipose cell size. J Clin Invest 1974; 53: 848-56.
17. Bary GA, Glennon JA, Salans LB, Horton ES, Danforth E Jr, Sims EA. Spontaneous and experimental human obesity: effects of diet and adipose cells size on lipolysis and liopogenesis Metabolism 1977; 26: 739-47.
18. Willa A. Hsueh, MD;1 Kathleen Wyne Renin-Angiotensin Aldosterone System in Diabetes and Hypertension J Clin Hypertens (Greenwich). 2011;13:224-237.
19. Kurukulasuriya LR, Stas S, Lastra G, et al. Hypertension in obesity. Endocrinol Metab Clin North Am. 2008; 37(3):647-662,
20. Nagel JM, Tietz AB, Goke B, et al. The effect of telmisartan on glucose and lipid metabolism in nondiabetic, insulin-resistant subjects. Metabolism.