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Metabolic Syndrome May be a Sign of Rapid Aging

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Abstract

The metabolic syndrome [MS] is a combination of the most dangerous heart attack risk factors. Several definitions have been suggested with the most acceptable being the IDF definition while the most recent is the Joint Interim statement. Irrespective of the definition used, the MS Several studies point to the metabolic syndrome as a potential target to avert aging.

Some predisposing conditions that increase in prevalence during aging are actually the indicators or determinants of the metabolic syndrome (MS). Besides, very relevant to promoting normal aging is the prevention and treatment of MS and cardiovascular disease [CVD].

From the Biochemical, pathophysiological and hormonal stand points, the metabolic syndrome can be considered as a sign of rapid aging.

It can thus be stated that the metabolic syndrome is a sign that the individual is aging faster. Preventing and treating this global time bomb should rekindle normal aging by decelerating rapid or premature aging.

Keywords: Metabolic syndrome; Rapid aging; Cardiovascular disease; Metabolic Syndrome definitions

List of Abbreviations

E.G.I.R: European Group for Study of Insulin Resistance; AHA: American Heart Association; AACE: American Association of Clinical Endocrinologists; IDF: International Diabetic Federation; JIS: Joint Initiative Statement; WHO: World Health Organization; NCEP/ATP-III: National Cholesterol Education Program-Adult Treatment Panel III; NA=Not applicable; Urinary Albumin Excretion Rate > =20 µg/min or Albumin: C reatinine ratio > =30 mg/g; MS: Metabolic Syndrome

Background

The 1988 Banting lecture given by Gerry Reaven introduced the concept of insulin resistance and later described "Syndrome X" which was later termed the metabolic syndrome. The Metabolic Syndrome [MS] is a cluster of the most dangerous coronary risk factors. These risk factors or determinants include central obesity, hyperinsulinemia, glucose intolerance, high triglycerides level, low "High Density Lipoproteins" (HDL), and hypertension. Added to these confirmed determinants are emerging determinants such as micro-albuminuria, uric acid and pro inflammatory cytokines [1]. Several definitions have been suggested by different organizations for the Metabolic Syndrome. The World Health Organization (WHO) in 1998, provided a definition of the metabolic syndrome as association of glucose intolerance with three or more other components [2]. In response, the European Group for the Study of Insulin Resistance countered with a modification of the WHO definition having insulin resistance as its hallmark [3] By 2001, the USA National Cholesterol Education Program (NCEP) released its own definition categorizing the risk factors as underlying, major and emerging [4]. Subsequently, the American Association of Clinical Endocrinologists considered the concept of insulin resistance as central [5]. The proliferation of definitions informed the need for a single unifying definition [6]. In the hope of accomplishing this, the International Diabetes Federation (IDF) proposed another definition for the metabolic syndrome, better suited for use in epidemiology studies and clinical practice, and which would allow for comparison between different population groups and the assessment of its relationship with various health outcomes [7]. In 2009, an additional definition of metabolic syndrome-a Joint Interim Statement was proposed by several organizations in an attempt to harmonize the definition of the metabolic syndrome [8] (Tables 1a and 1b). These determinants or components of metabolic syndrome occur together more frequently than expected by chance, and when grouped together they result in an increased risk for cardiovascular disease and diabetes mellitus [1,6]. The IDF definition of MS associates more strongly than the NCEP/ATP-III definition with the concept of sedentary lifestyle [9].

A lot of controversy surrounds the actual point an organism begin to grow old. It can be at conception or maturity and the aging process may constitute a process of evolution or involution [10,11] but some authors believe that aging being an inherently complex process is manifested within an organism at genetic, molecular, cellular, organ, and system levels [12]. Others hold that an individual is only young up to the age of 25 years, after which the human body enters a regressive mode [13]. Aging is associated with a loss of both muscle mass and the metabolic quality of skeletal muscle.

Salient features and obligatory criteria	Glucose intolerance, IGT or diabetes and/or insulin resistance* together with two or more of the following	Insulin resistance (defined as hyperinsulinaemia —top 25% of fasting insulin values among the non-diabetic population). Plus two of the following	Three or more of the following five risk factors
Fasting plasma glucose	≥6.1 mmol/l (110 mg/dl)	≥6.1 mmol/l (110 mg/dl) but non-diabetic	≥5.6 mmol/l (100 mg/dl)[a]
Blood pressure	≥140/90 mmHg	≥140/90 mmHg or treatment	≥130/≥85 mmHg
Triglycerides	Raised plasma triglycerides	>2.0 mmol/l (178 mg/dl)	≥1.7 mmol/l (150 mg/dl)
	≥1.7 mmol/l (150 mg/dl)	or treatment	
HDL-cholesterol	Men: <0.9 mmo1/1(35 mg/dl)	<1.0 mmo1/1(35 mg/d1)	Men: <1.03 mmo1/1(40 mg/d1)
	Women: <1.0 mmol/l (39 mg/dl)	or treatment	Women: <1.29 mmol/l (50 mg/dl)
Obesity	Men: waist–hip ratio >0.90	Men: waist circumference>94 cm	Men: Waist circumference > 92 cm
	Women: waist–hip ratio > 0.85 and/or BMI > 30 kg/m^2	Women: waist circumference ≥ 80 cm	Women: waist circumference > 88 cm
Microalbuminuria	Urinary albumin excretion rate ≥ 20 µg/min or albumin:creatinine ratio ≥ 30 mg/g	NA	NA

Tables 1a: The Different Metabolic Syndrome Definitions

Criteria	IDF (2005)	AHA (2004)	ACE/AACE)	JIS (2009)
Fasting Total cholesterol	≥5.2 mmol/l	NA	NA	NA
Fasting plasma glucose	≥5.6 mmol/l (100 mg/dl)	≥5.6 mmol/l (100 mg/dl)a	≥5.6 mmol/l (100 mg/dl)a	≥5.6 mmol/l (100 mg/dl)a
Blood pressure	≥130/85 mmHg	≥135/85 mmHg or treatment	≥ 130/ 85 mmHg	≥130/ 85 mmHg
Triglycerides	Raised plasma triglycerides ≥ 1.7 mmol/l (150 mg/dl) and/or triglyceride lowering drug	>1.7 mmol/l (150 mg/dl) or treatment and/or	≥1.7 mmol/l (150 mg/dl)	≥1.7 mmol/l (150 mg/dl)
HDL-cholesterol	Men: <0.9 mmol/l (35 mg/dl)	men < 1.03 mmol/l (40 mg/dl)	Men: <1.03 mmol/l (40 mg/dl)	Men: <1.00 mmol/l (39 mg/dl)
Obesity	Women:<1.0 mmol/l (39 mg/dl)	women < 1.29 mmol/l (50 mg/dl) or treatment	Women: <1.29 mmol/l (50 mg/dl)	Women: <1.3 mmol/l (50 mg/dl)
	Men: waist circumference ≥ 94 cm	Men: waist circumference ≥ 102 (40 inches)	Men: waist circumference > 102 cmb	Men: waist circumference ≥ 94 cm
	Women: waist circumference ≥ 80 cm	Women: waist circumference ≥ 88 cm(35 inches)	Women: waist circumference > 88 cm	Women: waist circumference ≥ 80 cm
Microalbuminuria	Urinary albumin excretion rate ≥ 20 µg/min or albumin:creatinine ratio ≥ 30 mg/g	NA	NA	NA

Table 1b: E.G.I.R: European Group for Study of Insulin Resistance; AHA: American Heart Association; AACE: American Association of Clinical Endocrinologists; IDF: International Diabetic Federation; JIS: Joint Initiative Statement; WHO: World Health Organization; NCEP/ATP-III: National Cholesterol Education Program-Adult Treatment Panel III; NA=Not applicable. Urinary albumin excretion rate > =20 µg/min or albumin:creatinine ratio > =30 mg/g.

Sarcopenia, the loss of muscle mass associated with aging, is a main cause of muscle weakness in old age and leads consequently to an increased risk for development of obesity-associated insulin resistance and type 2 diabetes mellitus [14,15].

Due to the metabolic consequences of reduced muscle mass, it is understood that normal aging and/or decreased physical activity may lead to a higher prevalence of metabolic disorders. Hormones and dietary habits play an important role in the pathogenesis of metabolic syndrome and aging. From the dietary standpoint, increased consumption of fish, low-fat dairy, fruits and vegetables and decreased consumption of butter, margarine, sweets, candies, soft drink [16] are all rejuvenating strategies in anti- aging medicine and also preventive strategies against the MS. Some authors reported a significant negative correlation between plasma Ghrelin, Insulin and leptin with significant

positive correlation between plasma insulin and leptin in obese and MS groups [17].

Menopause is associated with loss of ovarian functions and a reduction in endogenous estrogen leading to change in fats metabolism and increased fats deposit in adipose tissue [16]. This explains why postmenopausal women have a higher chance of suffering from the metabolic syndrome.

As a woman moves from pre- to post menopause, the emergence of many determinants of the metabolic syndrome becomes very apparent. Indicators of the MS such as raised insulin and glucose levels accompanied by a more artherogenic profile (raised LDL and triglycerides, low HDL) and finally a more pronounced abdominal obesity appear more likely in the post-menopausal women [18]. The emergence of these risk factors may be a direct result of ovarian failure or, alternatively, an indirect result of the metabolic consequences of central fat redistribution with estrogen deficiency. The increased prevalence of the metabolic syndrome with menopause may partially justify the apparent acceleration in CVD after menopause. Improved obesity, better glycemic control, normal blood pressure, satisfactory lipid profile, and enhanced bone mineral density all can be achieved by hormonal replacements to normalize testosterone (T) levels [19].

Although historically considered as a "normal" consequence of the aging process, benign prostatic hypertrophy [BPH/LUTS] now appears to be a preventable disorder of the elderly which can be prevented by healthy diet and appropriate level of physical activity [20]. Very relevant to promoting normal aging is the prevention and treatment of MS and CVD. Besides the prevalence of MS increase with age and accompanying lower hormone levels especially androgens, total cholesterol, and SHBG all of which can predict a higher incidence of MS [21].

Aging is characterized by pathological indicators such as increased lipo-peroxidation, generation of free radicals, increased peroxidation of nitric oxide [NO] to its toxic species, and increased oxidative stress which significantly alter the incidence of CVD [22]. Insulin resistance, changes in body composition, physiological declines in growth hormone (GH), insulin-like growth factor-1 (IGF-1), and sex steroids [23] are the key indicators of aging. Toxic reactive oxygen species (free radicals) liberated by the mitochondria and other cells often associated with MS contribute significantly to aging. Obesity, insulin resistance and ageing are all associated with impaired micro-vascular responses to insulin in skeletal muscle [24]. MS and depressive symptoms are independently related to CRP [25]. Aging is accelerated when metabolic and cardiovascular diseases (CVD) are present and the risk of these diseases increases with age. Many predisposing conditions which increase in prevalence during aging, such as obesity, insulin resistance, inflammation, changes in the activity of the hypothalamus-hypophysis suprarenal axis, stress and hypertension also contribute to increase prevalence of metabolic syndrome (MS) and CVD [26]. Metabolic abnormalities in MS are a risk factor for developing complications in the peri-operative period of patients scheduled for surgeries using the subarachnoid anesthesia technique [27]. 21 Cockayne syndrome (CS) is a rare disorder characterized by short stature and an appearance of premature aging. Diagnosis depends on presence of three features growth retardation, abnormal sensitivity to light (photosensitivity) and prematurely aged appearance (progeria). Features of this disorder include a failure to gain weight and grow at the expected rate, abnormally small head size (microcephaly), and impaired development of the nervous system. CS is an accelerated aging disorder whose pathogenesis is characterized by progressive neuro-degeneration caused by mutations in genes encoding the DNA repair proteins CS group A or B (CSA or CSB) [28].

Old age is an unpreventable physiological state and epidemiologically independent risk factor for chronic non-communicable diseases. Prevalence of chronic conditions especially among geriatric women are considered as the major causes of disability in the elderly population [29]. The consequences of diseases in later life have been judged predominantly through mortality, resulting in an emphasis on the fatal rather than the nonfatal disabling conditions [30].

The biochemical markers of cardiac disease [hyperglycemia, low HDL, high LDL, elevated uric acid and triglycerides] all constitute the biomarkers of aging as well as a central integrative role of neuro-inflammation in metabolic syndrome components ranging from obesity, glucose intolerance to cardiovascular dysfunctions has been reported [31]. Arteriosclerosis is a condition which can be associated to aging [32] and aging triggers signs of metabolic syndrome in rats [33]. Chronic inflammation is known to be associated with visceral obesity and insulin resistance which is characterized by production of abnormal adipocytokines such as tumor necrosis factor α, interleukin-1 (IL-1), IL-6, leptin, and adiponectin [34]. As the human being ages, the immune system undergoes a process of senescence accompanied by the increased production of proinflammatory cytokines and these inflammatory reactions can predispose to neuropsychiatric and neurodegenerative diseases [35]. Strategies that reduce age-related inflammation may improve the quality of life in older adults [36]. Chronic low-grade inflammation is mediated by adipocytokines (adipokine inflammation). This inflammation is modulated by dietary factors such as glucose and lipids can influence adipokine inflammation and consequently insulin resistance directly through their effects on secretion of adipocytokines (TNFα, IL6 and resistin) as well as indirectly through increases in endotoxin [37,38].

When the deleterious complex of diseases, disabilities and impairments of normal aging are minimized by controlling the blood pressure, blood glucose levels, blood lipids levels and the total body fat, then successful aging is ushered in. All these ensure that body functions are preserved until senescence makes a continued life impossible. Preventing or treating the determinants of the metabolic syndrome(abdominal obesity, hypertension, hyperglycemia, low HDL, high LDL, elevated uric acid, triglycerides and elevated levels of pro-inflammatory cytokines) not only improve the quality of life but also slows down the aging process.

Several studies point to the metabolic syndrome as potential targets to avert aging [31,33] and the MS is significantly associated with decreased cognitive dysfunction and higher frailty incidence, even though the causes of cognitive decline in diabetes is complex and multifactorial [34]. Some predisposing conditions that increase in prevalence during aging, such as obesity, insulin resistance, inflammation, changes in the activity of the hypothalamus-hypophysis suprarenal axis, stress and hypertension also contribute to increase prevalence of metabolic syndrome (MS) and CVD [32]. Actually, some of these predisposing factors are actually determinants or indicators of MS. Diet, exercise, hormonal enhancement, nutritional supplement, and stress reduction all constitute the five pillars of ant-aging medicine 26 as well as being the successful strategies to prevent the metabolic syndrome. Weight gain predisposing to overweight and or obesity, increased blood pressure, elevated levels of cholesterol, triglycerides and blood glucose are all accepted signs of aging as well as indicators or determinants of the metabolic syndrome [26]. Globalization and

modernization with its 24×7 society has wrongly degraded sleep both in quantity and quality to a requirement of diminished importance. With fast paced live being a global phenomenon, the attendant sleep deprivation can induce stress [27]. which can in turn predispose to obesity and eventually the metabolic syndrome. The presence of the MS in subjects below 40 years may be considered as a sign of premature aging. Preventing and treating MS and CVD would be useful in promoting normal aging [26]. Dietary intake is closely correlated with kidney damage in patients with metabolic syndrome and thus high protein intakes may be avoided [38]. It is imperative to implement programs to screen these risk factors or determinants of the MS by means of routine medical exams. Screening programs should be instituted at the community level for the early diagnosis, treatment of MS, and further regular monitoring of the treatment compliance to control diseases related morbidity and mortality. Medical personnel must not be left out of this preventive screening for MS as some studies [39] seem to suggest that the MS prevalence rate is also high among the medical personnel in hospital settings. Although some recent studies seem to suggest that MS is not linked to lower urinary tract infections (LUTI) in men [40], it is associated with a decline in muscle mass [41] especially as from middle age. The better knowledge of mechanisms linking MS to increased CVD prevalence has led to new predictive measures and to the study of different possible new therapeutic strategies in elderly patients with MS.

Conclusion

From the Biochemical, pathophysiological and hormonal stand points, the metabolic syndrome can be considered as a sign of rapid aging. Preventing and treating this global time bomb should rekindle normal aging by decelerating rapid or premature aging.

Competing Interest

I hereby declare that they were no competing interests.

Author's contribution

GKT conceived, prepared all the different versions of the draft and edited the manuscript.

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