



Editorial – referring to the article published on pp 595–604 of this issue

The Metabolic Syndrome and Erectile Dysfunction: Multiple Vascular Risk Factors and Hypogonadism

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In this issue Corona and colleagues conclude that erectile dysfunction (ED) is more severe in patients with the metabolic syndrome and associated with an increased incidence of hypogonadism [1]. The findings have important lifestyle, prevention, and therapeutic implications.

Unfortunately, the definition of the metabolic syndrome is not as precise as the name implies [2]. It is an umbrella term for a cluster of cardiovascular risk factors including type 2 diabetes, hyperlipidaemia, abdominal obesity, and hypertension secondary to “insulin resistance” and as a consequence, hyperinsulinaemia. Umbrellas protect us from the elements and there is a danger that in using a unifying label we may not address each element of risk optimally. The definition itself has been challenged by the American Diabetes Association and the European Association for the Study of Diabetes [2]. In a joint statement they conclude that “too much critically important information is missing to warrant its designation as a ‘syndrome.’” They encourage the treatment of all cardiovascular risk factors irrespective of whether the “metabolic syndrome” has been diagnosed. Although the National Cholesterol Education Program’s Adult Treatment Panel (ATP III), World Health Organization (WHO), and International Diabetes Federation (IDF) offer different definitions, there are similarities we can extract for day-to-day practical use [3–5]. The first is central obesity with a waist-to-hip ratio >0.90 in men and >0.85 in women, which translates to a waist circumference in European men of ≥ 94 cm

and ≥ 80 cm in women. In addition there should be any two of the following: raised triglyceride level >1.7 mmol/l, reduced high-density lipoprotein (HDL) cholesterol of <1.0 mmol/l in men and <1.3 mmol/l in women, blood pressure $>130/85$ mm Hg or treated blood pressure or raised fasting glucose ≥ 6.1 mmol/l (and possibly 5.6 mmol/l). Including diabetics and those with clinical cardiovascular disease seems pointless because their risk status is known so that they add little to the understanding of risk for those otherwise having features of the “metabolic syndrome.” Therefore, however we view these varying definitions, what we are talking about is increased vascular risk and, in turn, reducing it. The best philosophy is never to view a single vascular risk factor in isolation.

The patients in the study by Corona et al. had multiple risk factors with considerable central obesity. The questionnaire component reflecting the presence of organic ED showed a progressive increase as the number of metabolic syndrome components increased. In addition, there was a 3-fold increase in the prevalence of hypogonadism and as the number of criteria for the metabolic syndrome increased so did the incidence of hypogonadism. The major determinants of hypogonadism were abdominal obesity and hyperglycaemia, both of which have been previously connected [6,7]. Makhside et al., reviewing the literature from 1988 to 2004, reported a strong association between hypogonadism and “metabolic syndrome” [8]. They speculated that testosterone therapy might have a

role as a treatment option for the metabolic syndrome though no studies have been done to support this idea. I think we need to walk before we run, but the idea is attractive and worthy of study.

In Corona's study, the impact of ED is reflected in increased somatised anxiety—a process whereby an emotion is expressed or experienced as a physical symptom leading to a symptom being falsely considered to originate in a physical illness. Essentially, the individual has little or no insight into the problem, going from doctor to doctor seeking a physical diagnosis or explanation. In other words, ED is distressing and a cause of much unhappiness.

So what can be our practical take home messages:

- All vascular risk factors need to be measured in all patients with ED. They need to be addressed aggressively to reduce cardiovascular morbidity and mortality and perhaps benefit ED [9].
- Accumulative risk factors compatible with the “metabolic syndrome” increase the incidence of ED, but the diagnostic label should not lead to complacency about overall risk reduction.
- Lifestyle changes are essential as a form of prevention and management.
- Hypogonadism is associated with coronary artery disease and increased vascular risk as part of the metabolic syndrome. Testosterone should be measured routinely. Defining hypogonadism as a total testosterone level of <8 mmol/l assumes an absolute rather than relative diagnosis and this may need to be revisited as part of individualised management (some men may need to be more than just normal).

It is to be hoped Corona and colleagues will report back to us on the impact of testosterone

replacement therapy on their patients with ED and hypogonadism and any changes in vascular risk parameters observed in their study.

References

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