



Interrelation between blood pressure and diabetes

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It has been known for some time that certain comorbidities have the ability to exaggerate the severity of primary hypertension and display synergistic effects on target organ damage. Several interrelating phenotypes with primary hypertension have been described, of which the most common are the components of the metabolic syndrome i.e. insulin resistance with concomitant hyperinsulinemia, central obesity and dyslipidemia (1). The absence of cut-off values for the quantitative traits comprising the syndrome has obscured the advent of a clear-cut definition of the metabolic syndrome. Further complexity in relation to the definition of the metabolic syndrome comes from the fact that its components are reciprocally interrelated with incomplete knowledge of their individual contribution to the pathophysiology of the syndrome (2). Many study groups such as the World Health Organization (WHO), NCEP 2002 (National Cholesterol Education Program) and European group for the study of Insulin Resistance have suggested definitions of the metabolic syndrome. These definitions are fairly well correlated giving the prevalence of the metabolic syndrome in around 25% in the general population (3). It has been suggested that insulin resistance and compensatory hyperinsulinemia underlie the clustering of metabolic disturbances and that the syndrome itself is an important risk factor for cardiovascular disease (4).

In the clinical setting, obesity, hypertension and dyslipidemia are more commonly seen with type II diabetes. Commonly, derangements in fibrinolysis, coagulation and inflammation add to the cardiovascular risk of the metabolic syndrome (5). There is a large body of experimental evidence that insulin resistance and compensatory hyperinsulinemia are increased in patients with primary hypertension, and similar changes can be seen in first degree relatives of patients with primary hypertension (6). However, these studies have not been able to establish the causality of the observed link. It is likely that the elevated insulin levels cause the rise in blood pressure, as prospective studies have shown that a high insulin level, even within normal ranges, is a strong independent risk factor for developing subsequent primary hypertension (7). About 50% of hypertensive subjects are insulin resistant and it should not be obscured that even though insulin resistance and hyperinsulinemia

■ Implication for health policy/practice/research/medical education

It has been known for some time that certain comorbidities have the ability to exaggerate the severity of primary hypertension and display synergistic effects on target organ damage. Several interrelating phenotypes with primary hypertension have been described, of which the most common are the components of the metabolic syndrome i.e. insulin resistance with concomitant hyperinsulinemia, central obesity and dyslipidemia. The absence of cut-off values for the quantitative traits comprising the syndrome has obscured the advent of a clear-cut definition of the metabolic syndrome.

■ **Keywords:** Hypertension, Cardiovascular disease, Dietary effect

do not contribute to the aetiology of primary hypertension in some individuals, they do in others, most likely due to genetic differences in biomolecular pathways linking insulin to blood pressure (8). One such pathway, involving the serum glucocorticoid regulated kinase type 1 (SGK-1) gene, has recently been identified, thereby providing a link between hyperinsulinemia and over-activity of the epithelial sodium channel (ENaC) in the collecting ducts of the kidney (9). This pathway will be explained in more detail below. In a study of Mexican/Americans and non-Hispanic whites, 13.8% of obese subjects (body mass index greater than 30 kg/m²) were hypertensive, compared to 6.3% among nonobese subjects. The same study showed that hyperinsulinemia as a marker of insulin resistance increases the prevalence of primary hypertension from 6.9% in normoinsulinemic subjects to 13.4% in hyperinsulinemic subjects. In general, around 50% of type 2 diabetics display primary hypertension (9), which is considerably higher than in nondiabetic subjects, thus clearly showing that primary hypertension clusters among the components of the metabolic syndrome. Furthermore, it has been shown that subjects fulfilling the criteria for the metabolic syndrome have a greatly increased risk of cardiovascular mortality (10).

Authors' contribution

MAL completed the article. CMJN, SJK and HAL completed the draft.

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Ethical considerations

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