

L90

HYPERTENSIVE DISORDERS AND DIABETIC PREGNANCY

Jacob Bar, Moshe Hod, *Perinatal Division, Department of Obstetrics & Gynecology, Rabin Medical Center, Beilinson Campus, Petah Tikva, and, Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel*

Barker et al pioneered the idea that the epidemic of coronary heart disease in Western countries in the 20th century, which paradoxically coincided with improved standards of living and nutrition, originated in fetal life. An association of low birth weight with high risk of later ischemic heart disease and stroke, or impaired glucose tolerance and diabetes mellitus (DM) was found in other studies. Accordingly, low birth weight was also found to be associated with high blood pressure (BP) in childhood and adult life. In another study, Barker et al observed that the effects of impaired fetal growth are modified by subsequent growth. As such, individuals who were small at birth but became overweight in adulthood, were at the highest risk of heart disease and type 2 (non-insulin-dependent) DM, a physiological resistance to insulin action. This finding led to the second part of the hypothesis, the thrifty phenotype. The insulin resistance syndrome is characterized by a cluster of clinically recognizable physiological abnormalities, namely, glucose intolerance, high BP, and unfavorable lipid profile, all alterations induced by the compensatory hyperinsulinemia. It also involves biochemical abnormalities. Insulin resistance now appears to be the epidemiological link between high BP and obesity, both risk factors for developing cardiovascular disease later in life. The insulin resistance can induce hypertension include via mechanisms at the cellular, circulatory, and neurological levels, as well as via possible polygenic factors. Acquired or transient insulin resistance is associated with certain physical conditions, such as pregnancy, obesity, oral contraceptive use, and severe distress. Type 2, or non-insulin-dependent DM, is a state of increased insulin secretion owing to the physiological resistance of insulin action and lower than normal beta cell reserve. Diabetes in pregnancy, or gestational DM (GDM) may precede the clinical expression of type 2 DM in the nonpregnant state, even by several years. Preeclampsia and other hypertensive disorders, which are known to have a higher incidence in GDM, can be linked to increased insulin resistance. To understand the association between insulin resistance and hypertensive disorders in pregnancy, we first need to elucidate the role of insulin resistance in hypertensive disorders in the nonpregnant state. The pathogenesis of essential hypertension is multifactorial, involving complex interactions between endocrine, metabolic, and genetic factors. Obesity, aging, and diabetes can amplify genetic tendencies toward the clinical expression of the disorder.

Gestational Diabetes and Hypertensive Disorders: The study of both GDM and PIH has suffered from the lack of international consensus about classification, definitions, and nomenclature, leading to difficulties in comparing studies that used different diagnostic criteria. Nevertheless, epidemiological and physiological evidence suggests that GDM and PIH are etiologically distinct entities and that GDM is strongly associated with insulin resistance and glucose intolerance, whereas preeclampsia is probably not.

Pregestational diabetes and hypertensive complications

In most cases, pregestational diabetes refers to type 1 DM; the incidence in of type 1 DM in pregnancy ranges from 0.2-0.5%. These women make up a heterogeneous group in terms of duration of diabetes (White's classification), presence of hypertension, and end-organ damage, especially damage to the eye (retinopathy) and kidney (nephropathy). Pregnancy in women with type 1 diabetes is associated with increased risks of preeclampsia, intrauterine growth restriction (IUGR), neonatal morbidity, and perinatal mortality.