

Clinical Digest

ONLINE EDITION

WOMEN'S HEALTH

Insulin Resistance: Another Clue to Endometrial Cancer?

Obesity has been linked to endometrial cancer (EC), as has diabetes, but little is known about the role insulin resistance (IR) might play. In preclinical studies, IR and high circulating levels of insulin have potentiated the effect of estrogen on endometrial proliferation. Researchers from the University of Texas M.D. Anderson Cancer Center, Baylor College of Medicine, both in Houston, and University of Puerto Rico, conducted a multicenter study to determine the prevalence of IR in newly diagnosed EC patients.

Between 2005 and 2008, they enrolled 99 women, all patients with newly diagnosed EC, from 5 different hospitals and 1 private practice. Study participants were asked to com-

plete a structured, self-administered questionnaire that focused on demographics, menstrual and reproductive history, family history, and personal medical history. Medical records were reviewed, height and weight were measured, and body mass index (BMI) was calculated.

IR was identified in 66 participants, 30 of whom had a previous diagnosis of diabetes. In 36 women, the diagnosis was based on a low quantitative insulin sensitivity check index (QUICKI), which is calculated by the formula (1/[log fasting insulin + log fasting glucose]). More than half of the insulin-resistant women did not have diabetes and diabetes had not been previously diagnosed.

Although race and ethnicity were not significantly associated with increased IR, the researchers did note a high prevalence of IR (80%) among Hispanic women who had EC. The Hispanic women were significantly younger, more obese, and of lower social-economic status than the women of other ethnic groups.

Nationwide, the researchers note, 9% of incident cases of EC, diagnosed between 2002 and 2006, were in Hispanic women. Given the high prevalence of IR, as well as the young age of onset of EC among the Hispanic women in their study, the researchers urge targeted education and prevention measures for this population.

The researchers also point out that classic models of EC pathogenesis link obesity and related estrogen excess to low-grade, early-stage disease, but they didn't find such an association in this study. The finding is intriguing, they say; it may indicate that IR contributes to all grades of EC. They note that insulin is a growth factor; in vitro studies demonstrate a mitogenic effect on EC cell lines.

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