



Enigmas of a mother's heart

Species that cannot successfully reproduce and raise their young will not survive. Humans have evolved complex ways to ensure the survival of the mother so that her immature young can be cared for. Impressive mechanisms prevent the gravid woman from rejecting her immunologically foreign fetus and prevent a graft-vs-host reaction as fetal cells cross into the mother's circulation and populate some of her organs. The mother's body also undergoes many well-choreographed physiologic changes to accommodate and nourish the fetus and prepare for the stresses of birth.

We tend to take successful gestation for granted. We understand the adverse fetal effects of environmental and genetic influences and infection, as well as the effects of diseases such as chronic kidney disease, diabetes, and pulmonary hypertension. But some disorders, such as preeclampsia and the HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count associated with preeclampsia), dramatically and uniquely affect the apparently healthy pregnant woman and thus require prompt recognition and treatment. Yet, despite their severity, their pathophysiology eludes our full understanding, and they tend to be underrepresented in our internal medicine curricula.

In this issue (page 289), Ramaraj and Sorrell discuss another enigma of maternal-fetal medicine, peripartum cardiomyopathy—a condition with significant heterogeneity in outcome and, likely, several triggers. But the variability of recurrence in subsequent pregnancy (even if cardiac function recovers from the first episode) argues that peripartum cardiomyopathy is not simply the effect of the cardiovascular stress of pregnancy on occult left ventricular dysfunction. This variability of recurrence also argues against a primary autoimmune mechanism, as does the observation that other autoimmune diseases often go into remission during pregnancy (but may flare postpartum). The higher incidence in African Americans and particularly in Haitians argues for some genetic contribution, and yet the variable rate of recurrence also challenges “bad genes” as the sole explanation.

Although the authors discuss diagnostic tests, peripartum cardiomyopathy still primarily requires astute clinical recognition with exclusion of other causes of acute heart failure. Thus, despite its low prevalence in the United States, this potentially fatal condition is worth reviewing. It is too tempting to attribute the early signs and symptoms of heart failure (dyspnea, edema, fatigue) to “just” the late stages of pregnancy.

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