ANDROGEN EXCESS IN THE DIAGNOSIS OF POLYCYSTIC OVARY SYNDROME (PCOS)
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Controversy has existed for some time regarding the diagnosis of PCOS. In the US the diagnosis has been based on the findings of irregular cycles (oligo/anovulation) and hyperandrogenism, in the absence of another cause such as tumor or an adrenal enzymatic defect. This was not a consensus decision but emanated from a conference held at the NIH in 1989. It did not require a particular ultrasound morphology of the ovary, which has been considered a hallmark feature of the diagnosis, hence the name PCOS. The “NIH” definition also did not provide for women who may have ovulatory function to have PCOS, a fact which has been documented repeatedly. A conference was subsequently held in Rotterdam which attempted to reconcile many of these differences between the “NIH” criteria and what was considered an European definition which was highly influenced by the presence of polycystic morphology on ultrasound. A compromise definition was crafted, known as the Rotterdam criteria as a result of the ESHRE/ASRM conference. This definition requires any two of three distinctive features: irregular cycles, hyperandrogenism, and polycystic ovaries on ultrasound. Clearly a woman with “classic” PCOS will have all three features. However, the Rotterdam definition also allows for two other phenotypes: women with normal ovulatory cycles, but hyperandrogenism and polycystic ovaries (Ovulatory PCOS) and women who have irregular cycles and polycystic ovaries on ultrasound, but with no evidence of androgen excess/hyperandrogenism.

It is this latter phenotype which is still being actively debated. Some feel that from a pathophysiological standpoint, the presence of androgen excess is key. Others feel that some women with this phenotype may not have PCOS and merely have hypothalamic anovulation/amenorrhea. Even within the Androgen Excess Society, there is lack of uniformity of opinion on this point.

Two significant issues cloud these deliberations. First, from a practical standpoint, inaccuracies in current assay systems do not allow for a clear cut diagnosis of androgen excess quite often in the absence of skin manifestations. Secondly, androgen excess may sometimes be cryptic and only manifest with ovarian stimulation, for example. Clearly, however, the presence of androgen excess makes most of the traditional features of PCOS (insulin resistance/overweight or obesity) worse or more severe. Thus in the absence of documented androgen excess, if PCOS exists it is mild and therefore subtle. In this setting, does the naming of the phenotype as PCOS matter? Does it affect treatment? More discussion on these points will be forthcoming. However, from a practical standpoint, while the majority of women with this phenotype (but not all) probably have a mild form of PCOS, it is not likely to affect management, at least while they maintain these features. However these patients should not be included in studies which attempt to define the genetics or pathophysiology of PCOS.

References: