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Original article

Low levels of 25(OH)D and insulin-resistance: 2 unrelated features or a cause-effect in PCOS?

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SUMMARY

Background & aims: Recent investigations have identified low vitamin D status as a hypothetical mechanism of insulin-resistance in Polycystic Ovary Syndrome (PCOS). Instead, some authors supported the hypothesis that low vitamin D levels and insulin-resistance are 2 unrelated features of body size in PCOS. Hence, we aimed to explore the association of 25-hydroxyvitamin D (25(OH)D) with anthropometric, metabolic and hormonal features in PCOS.
Methods: We assessed the association of low 25(OH)D levels with endocrine parameters, insulin-sensitivity evaluated by hyperinsulinemic euglycemic clamp (HEC) and body composition measured by DEXA in 38 women affected by PCOS.
Results: Low 25(OH)D (25(OH)D $<$ 50 nmo/L) was detected in 37% of the entire cohort of patients. Body Mass Index (BMI), in particular total fat mass ($p < 0.001$), resulted to be the most predictor factor of 25(OH)D levels whereas Sex Hormone Binding Globulin (SHBG), Free Androgen Index (FAI), glucose uptake and fat free mass were not.
Conclusions: Our data demonstrated that in PCOS low 25(OH)D levels are significantly determined by the degree of adiposity.

1. Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder in the reproductive-age women that is characterized by hyperandrogenism, ovarian dysfunction and polycystic ovarian morphology.[1,2] In addition, previous studies reported that the majority of women with PCOS are affected by insulin-resistance and compensatory hyperinsulinaemia.[3] Several hypotheses have been challenged trying to elucidate the mechanism through which PCOS is associated with elevated risk to develop insulin-resistance; in part, this may be explained by the fact that PCOS is often associated with obesity that itself is usually accompanied by insulin-resistance.[4] However, a substantial number of women affected by PCOS are insulin-resistant, but not obese[5]: the reason because such women are also at greater risk of diabetes, independently of obesity, is still unsolved. A number of recent investigations have identified vitamin D as a possible explanation to bridge this gap: this hypothesis comes from accumulating evidence linking type 2 diabetes and insulin-resistance with poor vitamin D status.[6–8] Accordingly, some authors supported a role of vitamin D in the pathogenesis of insulin-resistance associated with PCOS, independently of the body mass index.[9,10] However, in all these studies the evaluation of insulin-sensitivity was performed using indirect indices of insulin-resistance derived from fasting values of insulin and glucose that mostly reflect hepatic insulin-sensitivity, supplying a partial evaluation of insulin-resistance that is also caused by peripheral reduction of glucose uptake in PCOS.[11] Thus, the exact mechanism through which vitamin D deficiency may be related with the development of insulin-resistance in PCOS has not yet been fully elucidated; moreover, since several evidence suggested that vitamin D deficiency occurs commonly in obese PCOS patients,[12,13] it also needs to be hypothesized that vitamin D might be trapped by adipose tissue thanks to its lipophilic property thus, concluding that vitamin D deficiency and insulin-resistance might be two unrelated features in PCOS. Therefore, the still opened question remain whether vitamin D deficiency is conferred by obesity and it is unrelated with insulin-resistance or if vitamin D

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