VITAMIN D
UpDates



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# EDITORIAL

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Dear Colleagues,
In this issue you will find some updates on the possible role of vitamin D in chronic kidney disease and certain gynaecological conditions. Note that in both articles the expert authors start out by highlighting how common it is to also find vitamin D deficiency in these pathological conditions. In the case of chronic kidney disease, they attribute this deficit to reduced nutritional intake secondary to typical dietary restrictions, frequent associated gastrointestinal disorders, and reduced exposure to sunlight, secondary to the disability.
It is also pointed out that in this condition, native vitamin D deficiency is compounded by impaired calcitriol synthesis, resulting in altered mineral and bone metabolism (Chronic Kidney Disease-Mineral Bone Disorder, CKD-MBD) characterised by a state of secondary hyperparathyroidism, which, though initially "adaptive" subsequently becomes "maladaptive" if not corrected by adequate vitamin D supplementation.
The observation that, even in patients with advanced chronic kidney disease, requiring dialysis, cholecalciferol administration is associated with an increase in calcitriol synthesis, is interesting, since it demonstrates that there is also extrarenal production of calcitriol, even at the level of the same parathyroid glands.
Although the subject has been debated, current guidelines suggest that supplementation with native vitamin D (cholecalciferol or ergocalciferol) be used, especially to prevent the onset or progression of hyperparathyroidism, perhaps achieving serum 25(OH)D levels well above 30 ng/mL in these patients, which would be preferable.
Active vitamin D metabolites should be reserved for the more advanced stages of chronic kidney disease, when there are high serum parathormone levels, despite adequate 25(OH) D levels. It should not be forgotten that the use of these metabolites may be associated with hypercalcaemia, hyperphosphatemia, altered FGF-23 levels and excessive reduction in PTH levels such that the risk of adynamic bone disease is increased.
Even when active vitamin D metabolites should be used, it would be wise, in any case, to ensure supplementation with native vitamin D, given its extrarenal physiological effects and presumed extra-skeletal benefits.

For example, how about receptors, vitamin D-modulated genes and vitamin D-activating enzymes in different tissues, including those of the reproductive tract?
Have you noticed how much new literature there is in our usual bibliographic update on obstetrics and gynaecology?
The authors of the other article in this issue point out that genetic polymorphisms of the specific vitamin D receptor (VDR) have been associated with different levels of sex hormones and that the addition of vitamin D to granulosa cells is able to increase their synthesis. This could well justify correlations observed between vitamin D deficiency and menstrual cycle disorders or polycystic ovary syndrome, characterised by oligo-anovulation, clinical and/or biochemical

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signs of hyperandrogenism and polycystic ovary morphology.
This could also justify the positive effects observed with supplementation, especially if daily, of patients with ovarian polycystosis, in terms of infertility and the correction of certain typical associated metabolic alterations, including hyperinsulinism, dyslipidaemia and chronic inflammatory status.
All good reasons not to neglect a vitamin D status assessment and a potential opportunity for supplementation in these patients as well.
What are your thoughts? Happy reading!