CORRESPONDENCE

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Response to comment on "Hypogonadotrophic hypogonadism due to a mutation in the luteinizing hormone β–subunit gene"

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We thank Dr. Valdes-Socin and colleagues for the instructive comments on our case report [1] and for sharing their experience on induction of puberty and fertility in male patients with the very rare disease that is under discussion. We agree that the best initial therapeutic strategy for young males with luteinizing hormone (LH) deficiency is administration of human chorionic gonadotropin (hCG) or recombinant LH; these treatments improve spermatogenesis and maximize fertility potential [2]. In patients with hypogonadotrophic hypogonadism (HH), low-dose, intramuscular injections of testosterone have been the most commonly used regimen to induce puberty in young males who do not wish to be fertile in the near future. Gonadotropin treatment is physiological in nature but can be complex (requiring frequent injections) and thus is less commonly used than testosterone treatment. A recent, prospective multicenter study on adolescents with HH found that gonadotropins successfully induced both testicular growth and spermatogenesis, irrespective of prior full-dose testosterone replacement for up to 5.7 years [3]. However, some evidence suggests that prior long-term exposure to testosterone may negatively impact fertility potential of these patients. On the other hand, induction of puberty via gonadotropin injection may improve fertility potential of these patients. Although we used low-dose testosterone to induce pubertal changes in our present case (a convenient procedure complying with the wishes of the patient), we agree that the use of hCG or recombinant LH to induce puberty maximizes the testicular response and future potential fertility in patients with isolated LH deficiency.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

REFERENCES

1. Song JW, Hwang HJ, Lee CM, et al. Hypogonadotrophic hypogonadism due to a mutation in the luteinizing hormone β-subunit gene. Korean J Intern Med 2017 Jan 16 [Epub]. https://doi.org/10.3904/kjim.2015.373.
2. Potorac I, Rivero-Muller A, Trehan A, et al. A vital region for human glycoprotein hormone trafficking revealed by an LHB mutation. J Endocrinol 2016;231:197-207.
3. Rohayem J, Hauffa BP, Zacharin M, Kliesch S, Zitzmann M; German Adolescent Hypogonadotropic Hypogonadism Study Group. Testicular growth and spermatogenesis: new goals for pubertal hormone replacement in boys with hypogonadotropic hypogonadism? A multicentre prospective study of hCG/rFSH treatment outcomes during adolescence. Clin Endocrinol (Oxf) 2017;86:75-87.

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