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Kallmann Syndrome with Preserved Olfactory Bulb Morphology: A Rare Diagnostic Challenge in a Young Male

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Abstract

Kallmann syndrome (KS) is a rare genetic disorder (1:4,000-10,000, predominantly in males) marked by hypogonadotropic hypogonadism and anosmia due to defective GnRH neuron migration. Mutations in KAL1, FGFR1, and other genes are implicated. A 20-year-old male presented with absent puberty and lifelong anosmia. He had eunuchoid habitus, micropenis, gynecomastia, small bilateral testes, Tanner stage I genitalia, and absent axillary/facial hair. Hormonal profile confirmed hypogonadotropic hypogonadism (testosterone 30.2 ng/dL, LH 0.24 mIU/mL, FSH 1.68 mIU/mL) with normal prolactin and thyroid function. Semen analysis showed azoospermia, and karyotype was 46,XY. Scrotal USG demonstrated bilateral testicular atrophy (R: 2.2 × 1.0 cm; L: 1.7 × 0.9 cm). Abdominal USG was normal. Brain MRI revealed a normal hypothalamic-pituitary axis with structurally preserved olfactory bulbs. The patient received intramuscular testosterone decanoate (250 mg/mL) every 21 days, gradually increased from half to one ampoule. After four months, testosterone rose to 289 ng/dL, FSH increased slightly (2.03 mIU/mL), and LH remained suppressed (0.40 mIU/mL). This case highlights the diagnostic challenge of KS, with the classic triad present despite normal MRI olfactory bulbs. Diagnosis was confirmed through clinical, hormonal, cytogenetic, and radiological evaluation, and testosterone therapy promoted endocrine recovery and initiation of secondary sexual characteristics. *[J Assoc Clin Endocrinol Diabetol Bangladesh, 2025;4(Suppl 1): S62]*

Keywords: Kallmann syndrome, Hypogonadotropic hypogonadism, Anosmia, Gynecomastia, Testicular atrophy

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