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INTERESTING IMAGE

Incidentally Detected Juvenile-Pattern Bone Scintigraphy in a Young Man with Kallmann's Syndrome

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We report a case of incidentally detected juvenile-pattern bone scintigraphy in a 30-year-old male with Kallmann's syndrome. He had osteoporosis, and complained back and chest wall pain. Bone scintigraphy showed mild growth plate uptake in long bones, and multiple focal uptakes in bilateral costochondral junctions. Although he had been receiving hormone replacement therapy since his early teens, the treatment was insufficient because he failed to visit the clinic regularly. We assume that his insufficient hormone replacement resulted in the juvenile-pattern bone scintigraphy.

A 30-year-old man underwent Tc-99m dicarboxypropane diphosphonate bone scintigraphy to evaluate the cause of back and chest wall pain. He had been receiving testosterone replacement therapy since being diagnosed with Kallmann's syndrome in his teens. Kallmann's syndrome is a genetic condition presenting several symptoms of hypogonadotrophic hypogonadism [1]. He had typical Kallmann's symptom with anosmia. Mild growth plate uptake was detected in both humeral heads, the distal femur, and the proximal tibia, and focal uptakes were noted in the bilateral costochondral junctions on bone scintigraphy (Fig. 1), although male epiphyseal growth plates of most bones normally close by the age of 18–20 years [2, 3]. These uptake patterns are normally seen during late adolescence [4]. At the time of bone scintigraphy, his hormone assays revealed a luteinizing hormone level of 0.4 mIU/m (reference, 0.8–10.0 mIU/m), a follicle-stimulating hormone level of 0.0 mIU/m (reference, 0.9–8.9 mIU/m), and a testosterone level of 2.72 ng/ml (reference, 2.45–18.36 ng/ml). Although his testosterone level was within normal limit at that time, he has not had treatment regularly. Thus, his testosterone level was very unsteady with wide variation (range, <0.2–8.85). Sex steroids, both estrogen and testosterone, are essential controllers of bone growth and maturation. In both men and women, estrogen is needed for the acquiring of maximal peak bone mass [5], and testosterone plays an important role not only in stimulating bone growth and maturation but also in increasing bone density and strength [6]. Delay of skeletal maturation due to lack of testosterone results from the hypogonadotropic hypogonadism related with Kallmann's syndrome [7]. Also, the hypogonadotropic hypogonadism can be a cause for unsuccessful closure of epiphyseal growth zones at the proper time; thus, the lengths of the long bones are increased. Generalized osteoporosis is also present in Kallmann's syndrome [8]. His bone mineral densitometry was below the expected range for age in both femur wards (Z-score, -2.9) and lumbar spine (Z-score, -2.7). Although there was no demonstrable focal

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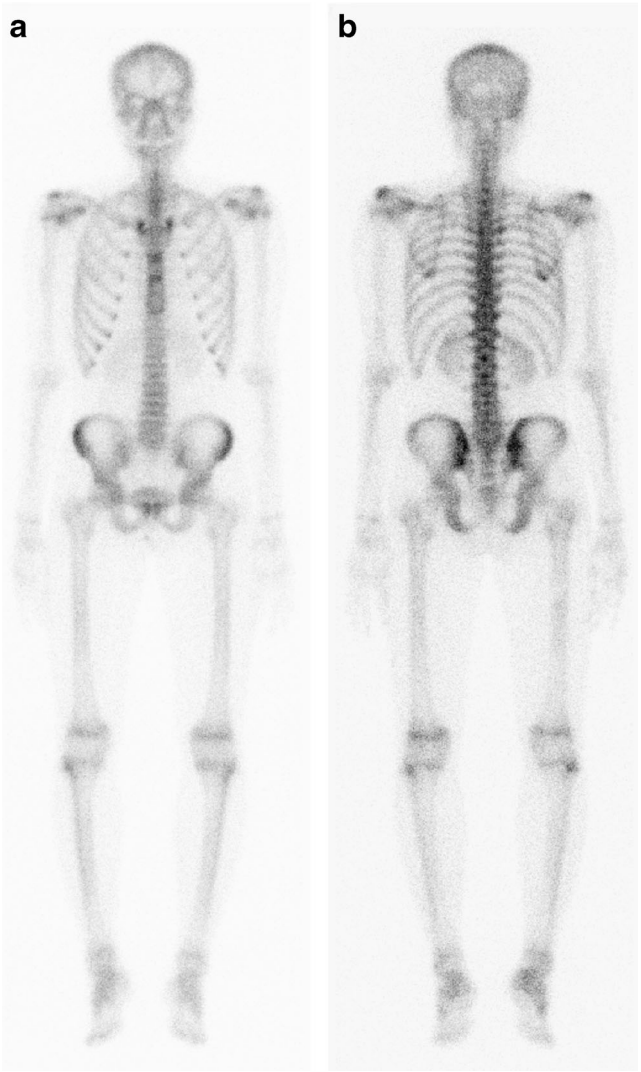

Fig. 1 Bone scintigraphy of a 30-year-old man with Kallmann's syndrome. **a** Anterior; **b** posterior

uptake suggesting fracture on his bone scintigraphy, we incidentally detected the juvenile pattern bone scintigraphy in the adult. We postulate that his insufficient hormone replacement resulted in the juvenile-pattern bone scintigraphy.

Conflict of Interest Soo Hyun Kwon, Yoon-Sok Chung, Dong Hyun Lee, Kyung-Sook Jo, Young-Sil An, Joon-Kee Yoon, and Su Jin Lee declare that they have no conflict of interest.

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