Editorial

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Commentary on "The association between idiopathic scoliosis and growth hormone treatment in short children"

Young Suk Shim

Department of Pediatrics, Ajou University Hospital, Ajou University School of Medicine, Suwon, Korea

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Address for correspondence:
Young Suk Shim
Department of Pediatrics, Ajou University Hospital, Ajou University School of Medicine, 164 World Cup-ro, Yeongtong-gu, Suwon 16499, Korea
Email: royjays@aumc.ac.kr
https://orcid.org/0000-0002-0850-5279

The first recombinant human growth hormone (rhGH) was approved for the treatment of growth hormone deficiency (GHD) in childhood by the Food and Drug Administration of the United States of America in 1985. It has been approved for use in several growth disorders including GHD, Turner syndrome, chronic renal failure, small for gestational age (SGA), Prader-Willi syndrome, Noonan syndrome, SHOX deficiency, idiopathic short stature (ISS), achondroplasia, short bowel syndrome and human immunodeficiency virus wasting syndrome.[1] Although rhGH therapy is well established to be safe and tolerable, some concerns about adverse effects remain.

Idiopathic scoliosis, which is defined as the development of spinal curvature of a minimum of 10°, is the most common type of scoliosis, affecting 2%–4% of children and adolescents, mainly those between 10 and 18 years of age.[2] In the general population, idiopathic scoliosis has been suggested to have both congenital and developmental components including genetic factors, estrogen, melatonin, diet, and exercise.[3] The velocity of spinal growth is a well-documented risk factor for idiopathic scoliosis.[4] Because the onset and progression of scoliosis are closely associated with growth spurts, scoliosis inevitably remains an issue during rhGH treatment. A few studies have demonstrated the link between rhGH treatment and scoliosis. The first report of growth hormone in relation to scoliosis progression described a Swedish male case in 1978.[5] Wang et al.[6] suggested that growth hormone may increase the risk of progression of scoliosis based on a study of 250 children being treated with growth hormone.

A recent study demonstrated that Cobb angle increased by 1.0° (P<0.001) per year in a rhGH patient group, whereas there was no significant annual change in the control group (P=0.496).[7] However, most studies regarding the potential relationship between rhGH treatment and scoliosis development or progression were conducted in a small sample,[5-7] and the impact of GH treatment on scoliosis remains controversial. Other studies indicated that the potential risk for scoliosis development during rhGH treatment is caused by rapid growth velocity rather than a direct effect of rhGH. One study suggested that about 4% of children with ISS who received rhGH therapy developed scoliosis, and this incidence was concordant with that of a general population without rhGH treatment.[8] A study by Park et al.[9] also reported that rhGH therapy was not associated with development or aggravation of idiopathic scoliosis, with no significant differences in average Cobb angle (6.2° vs. 6.1°, P=0.842) or prevalence of scoliosis (9.7% vs. 13.3%, P=0.481) before and after one year of GH treatment in 113 subjects who were diagnosed with GHD, SGA, or ISS.

Recently, rhGH treatment is being attempted at a younger age and corresponding skeletal immaturity. It is notable that skeletal immaturity is one of the risk factors associated with scoliosis progression.[10] Therefore, such patients are at high risk for development or progression of scoliosis. Although rhGH may not be directly related to scoliosis development and progression, patients receiving rhGH treatment who are expected to undergo growth spurts should be carefully monitored for early detection of scoliosis in an effort to prevent progression.

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