her genetic potential (mother’s height 159.5 cm, father’s height 176 cm, mid parental height 162.5 cm)

Conclusion Topical ocular administration of corticosteroid preparations, although rarely, may lead to the development of IAI. Growth suppression due to corticosteroid administration may occur in children without other symptoms of Cushing syndrome. Growth monitoring is required in all children receiving long-term topical corticosteroid therapy, and discontinuation of therapy should be gradual due to the possibility of developing of an adrenal crisis.

Introducing 46, XY partial gonadal dysgenesis (PGD) is a disorder of sex development characterised by an incomplete testicular development (dysgenetic gonad) which results in incomplete virilisation of external genitalia in utero and partial involution of Müllerian ducts in individuals whose karyotype is 46,XY.

Case Report A 14-year old girl was admitted due to primary amenorrhoea. A physical examination showed: BW 66 kg (88. c.), BH 156.7 cm (22. c.), BMI 24.9 kg/m2 (94. c.), axillary and pubic hair (Tanner stage 4), the lack of breast development (Tanner stage 1), clitoromegaly/micropenis (length of 3 cm) with the urethral opening at its tip, labial fusion, no palpable gonads; the rest of the physical examination was without abnormalities. She referred to herself as a female.

The results of the diagnostic evaluation: high gonadotropin levels – LH (11.6 IU/L) and FSH (58.6 IU/L), low levels of sex hormones – testosterone (0.6 nmol/L) and oestradiol (<32 nmol/L), low AMH (<0.21 nmol/L) and normal androstenedione (4.3 nmol/L) and 17-OHP (2.1 nmol/L). Karyotype was 46,XY with partial duplication Xp ((46,XY dup(X)(p11.4p22.1)). There was no increase in plasma testosterone (0.8-1.3), normal concentrations of alpha-fetoprotein, DHEAS, androstenedione and 17-OHP, immeasurably low concentrations of LH and FSH. Bone age assessment by Greulich-Pyle atlas was 13.5 years. Brain MRI: expansive lesion in the pineal region with radio-morphological characteristics of non-germinomatous germ cell tumor (NGGCT) – choriocarcinoma (according to laboratory findings). After chemo and radiotherapy remnants of the tumor were surgically removed (histopathology of previously irradiated tissue was noninformative).

Conclusion From our case, we can conclude that in patients with PGD, it is important to establish the diagnosis as soon as possible regarding the malignant potential of the dysgenetic gonads and the need of prophylactic gonadectomy.

INTRODUCTION

We present a boy with peripheral precocious puberty caused by germ cell tumor of the central nervous system. Our patient presented at the age of 10.5 years with accelerated growth and progressive virilization in the preceding 6 months (he gained about 11 cm in height, his muscle mass increased significantly, accelerated genital development was noticed, as well as acne occurrence and greasy hair). There were no headaches or visual disturbances, and he did not complain of frequent urination. Physical examination: BW 52 kg (96th ct.), BH 152 cm (93rd ct.), prominent muscles, genitals stage 4 on Tanner scale. Laboratory findings: increased concentrations of ßhCG (811 IU/L; ref. <5) and total testosterone (82.2 nmol/L; ref. 0.8-1.3), normal concentrations of alpha-fetoprotein, DHEAS, androstenedione and 17-OHP, immeasurably low concentrations of LH and FSH. Bone age assessment by Greulich-Pyle atlas was 13.5 years. Brain MRI: expansive lesion in the pineal region with radio-morphological characteristics of non-germinomatous germ cell tumor (NGGCT) – choriocarcinoma (according to laboratory findings). After chemo and radiotherapy remnants of the tumor were surgically removed (histopathology of previously irradiated tissue was noninformative).

Most cases of precocious puberty are seen in girls, in whom it is usually idiopathic. In boys, however, it is often caused by an underlying disease which is why it should always be taken seriously and investigated in a timely manner.