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POSTER COMMUNICATION · ENDOCRINOLOGY & DIABETOLOGY

From Growth failure to Gait failure: Arnold-Chiari 1 malformation in a teenager

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KEY MESSAGES

1. Arnold-Chiari type I malformation can cause growth failure and delayed puberty due to hypothalamic-pituitary axis involvement.
2. Early recognition of endocrine abnormalities in CM-I is critical for optimizing patient outcomes.
3. Multidisciplinary management is essential for accurate diagnosis and prevention of long-term complications.

ABSTRACT

Introduction. Arnold-Chiari type I malformation (CM-I) is a congenital anomaly of the craniovertebral junction characterized by caudal herniation of the cerebellar tonsils through the foramen magnum. It is classically associated with occipital headache, ataxia, and pyramidal tract signs, but its association with endocrine dysfunction remains underrecognized. Growth retardation and pubertal delay, although uncommon, may occur due to hypothalamic-pituitary axis involvement secondary to chronic intracranial pressure alterations or brainstem compression. Early recognition of endocrine manifestations in CM-I is critical for optimizing patient outcomes.

Materials and methods. A 17-year-old male, third child of non-consanguineous parents, presented with progressive growth failure for 6–8 years and difficulty walking for 4 years. Growth retardation became evident at 10 years despite normal appetite and school performance. Symptoms included chronic occipital headaches, blurred vision, intermittent dizziness, and progressive gait disturbance. Examination revealed disproportionate short stature (height 147 cm, arm span 161 cm, SDS -2.6), round facial features, scanty facial hair, under-pitched voice, delayed puberty (Tanner stage P2), left testicular volume 10 ml, right undescended testis palpable in inguinal region, dysarthria, tongue wasting with fasciculations, bilateral nystagmus, exaggerated deep tendon reflexes, extensor plantar responses, ankle clonus, and wide-based ataxic gait. Biochemical evaluation confirmed growth hormone deficiency; other pituitary hormones were normal. MRI showed foramen magnum stenosis, cervicomedullary compression, and dysplastic occipital condyles consistent with CM-1.

Results. Radiological bone age was 12-14 years, not corresponding with chronological age. Hormonal tests showed low TSH (1.17 mIU/L), normal FT4 (13 pmol/L), testosterone 7.71 nmol/L, ACTH 22 pg/ml, serum cortisol 317.6 nmol/L, FSH 4.27 U/L, LH 2.64 U/L. GnRH stimulation test showed LH increase from 2.64 to 26.83 mIU/ml. Short synacthen test showed basal cortisol 317.6 nmol/L and post-stimulation cortisol 1287 nmol/L. Growth hormone stimulation test showed basal GH 1.07 ng/ml and post-stimulation GH 3.41 ng/ml. IGF-1 was 250 ng/ml. MRI of pituitary and cervical spine confirmed structural abnormalities consistent with CM-1.

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ABSTRACT (CONTINUED)

Conclusion. CM-I typically presents with neurological manifestations; endocrine abnormalities are rare but important. Growth hormone deficiency and delayed puberty may result from hypothalamic-pituitary axis dysfunction secondary to cervicomedullary compression and disrupted CSF dynamics. Coexistence of short stature and delayed puberty highlights potential endocrine implications of structural brain anomalies. Early recognition of CM-I in patients with unexplained growth failure and neurological features is crucial. A multidisciplinary approach ensures accurate diagnosis, timely intervention, and prevention of long-term neuroendocrine sequelae.

POSTER



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