Coexisting Prolactin-Secreting microadenma and Type 1 Diabetes in an Adolescent Girl: Case Report

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Sixteen-year-old female patient diagnosed as type 1 diabetes at the age of 12 presented with primary amenorrhea, arrested puberty and galactorrhea. There was no history of headaches. At age of 16, her height was 161.5 cm (50 p) and weight was 49.5 kg (25 p). She had Tanner 3 pubic and axillary hair and breast development. She had galactorrhea. Prolactine levels were persistently elevated. Hypophyis MRI detected a low intensity mass measured approximately 7-8 mm in length expanding the adenohypophyis. She was diagnosed as microprolactinoma. She was begun on bromocriptine therapy. There have been no reports of patients with type 1 diabetes with symptoms of prolactinoma. In this paper we report a case with type 1 diabetes and prolactinoma.

Key words: Prolactinoma, type 1 diabetes,

Introduction

Pituitary adenomas occurring during adolescence and childhood are rare, prolactinoma being the most common. The most frequent symptoms are menstrual irregularities and galacthorrhea and gynecomastia in girls. Short stature and arrested puberty may be a symptom in both sexes (1). Some prolactin-secreting tumors lead to retardation of growth hormone as they diminish the pulstile output of growth hormone from pressure effects (2). Recently growth hormone producing pituitary adenoma was described with type 1 diabetes in an adult patient (3). There have been no reports of patients with type 1 diabetes with symptoms of prolactinoma. In this paper we report a case with type 1 Diabetes and prolactinoma.

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Case Report

Sixteen-year-old female patient diagnosed as type 1 diabetes at the age of 12 presented with primary amenorrhea, arrested puberty and galactorrhea. There was no history of headaches.

Her puberty degree was Tanner 3 when she admitted with hyperglycemia at the age of 12. Her height was 159 cm (50 p) and weight was 49 kg (25 p). She had Tanner 3 pubic and breast development Bone age was 13 years. She had achieved full remission with intensive insulin therapy in four weeks and was included into the immunotherapy program. After cessation of methylprednisolone insulin requirement decreased. She was followed without insulin therapy for 20 months. Blood glucose was normal and HbA₁ C levels were between 5.7-7.5% during this period. Hyperglycemia was detected at the end of 20 months and insulir therapy was initiated. At age of 16, her height was 161.5 cm. (50 p) and weight was 49.5 kg. (25 p). She had Tanner 3 pubic and axillary hair and breast development. She had galactorrhea. Fasting serum glucose was 104 mg/dl and HbA1 C was 4.7%. Bone age was 15 years. Thyroid function tests were normal. FSH, LH and E₂ levels were 5.2, 3.4 IU/l and 19.7 pg/ml respectively. Prolactine levels

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were persistently elevated (137-150 ng/ml). Pelvic ultrasound revealed normal uterus and ovaries. Hypophyis MRI detected a low intensity mass measured approximately 7-8 mm in length expanding the adenohypophyis. She was diagnosed as microprolactinoma. She was begun on bromocriptine therapy (initial dose 7.5 ng/day; increased to a daily dose of 10 ng/day) with a decline in the prolactine levels from 81 ng/ml to 40 ng/ml associated with pubertal progress on the 6h month of therapy.

Discussion

Arrested puberty in diabetic adolescents is usually considered to be due to diabetes mellitus. However, unrelated factors with diabetes may occasionally produce arrested puberty or growth retardation.

In general, pituitary adenomas are tumors of adults. They account for approximately 10% of all primary intracranial neoplasms and one third to one half of all sella/jukstasellar masses. In contrast pituitary adenomas are rare in children and account for only approximately 1% of pediatric intracranial masses and most adenomas are diagnosed in girls between 9-13 years of age. These are usually adrenocorticotrop hormone or prolactin secreting tumors (sperling).

Hyperprolactinemic states are associated with mild glucose intolerance; hovewer the difference is of only marginal statistical significance when compared with controls. Glucose intolerance and hyperinsulinism improves after treatment with bromocriptine (joslin). Although hyperprolactinemic our patient's HBA₁c levels did not change and she did not show any

signs of insulin resistance prior to bromocriptine therapy.

Some diabetic adult patients have presented with impotence due to prolactinoma (6,7). An adenoma is not reported in the literature up to date in an adolescent patient with IDDM. This state is evaluated as a coincidence. No side effect of the therapy and metabolic effect was observed in the follow up of the patient.

References

- Mindermann T, Wilson CB. Pituitary adenomas in childhood and adolescence. J Ped Endocrinol Metab 8: 79-83, 1995.
- Laws ER. Brain tumors affecting growth and development.
 In: Brook GD and Hindmarsh PC eds. Clinical Pediatric Endocrinology 4th ed. London: Blackwell Science, 2001: 253-258
- Otsuka F, Ogura T, Yamauchi T, Shikata K, Kageyama J, Makino H. IDDM accompanied by a growth hormoneproducing pituitary adenoma. *Diabetes Care* 20: 1838-41, 1997.
- Pollock AN, Towbin RB, Charron M, Meza MP. Imaging in Pediatric Endocrine Disorders. In: Sperling MA ed. Pediatric Endocrinology 2nd ed. Pennsylvania: Saunders, 2002: 725-756.
- 5. Gand OM. Secondary forms of diabetes. In: Kahn CR and Weir GC eds. Joslin's Diabetes Mellitus 13th ed. Pennsylvania: Lea and Febiger, 1994: 300-350.
- 6. Shakir KM. Impotance as a presenting symptom of presumed prolactinoma in a petient with diabetes mellitus. *Diabetes Care* **6:** 510-1, 1983.
- Lester E, Woodroffe FJ, Smith RL. Prolactin and impotence in diabetes mellitus. Ann Clin Biochem18: 6-8, 1981.