LETTER TO THE EDITOR

Serial T1-weighted magnetic resonance imaging changes in a patient with central diabetes insipidus, possibly due to lymphocytic infundibuloneurohypophysitis

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Central diabetes insipidus (CDI), characterized by polydipsia and polyuria due to vasopressin deficiency, is familial, idiopathic or secondary. Idiopathic CDI, which accounts for 10% to 30% of cases of CDI (1), is characterized by selective hypofunction of the hypothalamic–neurohypophysial system. Idiopathic CDI could be an autoimmune disorder (2). Imura *et al.* (3) studied the processes of idiopathic CDI by magnetic resonance imaging (MRI), found abnormalities in the pituitary stalk and neurohypophysis, and performed biopsies that demonstrated lymphocytic inflammation. Their cases were diagnosed with CDI due to lymphocytic infundibuloneurohypophysitis. Their study provided new insight into the pathogenesis of CDI. However, they did not demonstrate the serial changes by MRI. We demonstrate the serial T1-weighted MRI changes in a 52-year-old woman with CDI possibly due to lymphocytic infundibuloneurohypophysitis. The initial MRI findings on admission were prominent pituitary-stalk thickening and neurohypophysial enlargement. These findings suggested that the patient had lymphocytic infundibuloneurohypophysitis (3–5). The serial T1-weighted MRI changes which occurred over the years were (i) prominent pituitary-stalk thickening and neurohypophysial enlargement, (ii) their improvement, and finally (iii) empty sella. The MRI changes are associated with the clinical course of the disease. Steroids were not administered to the patient; she recovered from CDI spontaneously. This is the first report to demonstrate the natural course of serial MRI changes in a patient with CDI possibly due to lymphocytic infundibuloneurohypophysitis.

A 52-year-old woman had been well until 27 October 2002, when she began to have extreme thirst and excessive urine output. She visited us on 2 December 2002. She had extreme thirst and had started to drink more than 2 liters water a day. She had decreased anti-diuretic hormone (ADH) levels (0.3 ng/l, reference range 0.3–3.5 ng/l) with a low urine osmolality of 100 mOsm/kg, high plasma osmolality of 310 mOsm/kg (reference range 278–305 mOsm/kg), and a high serum

sodium (Na) level (147 mmol/l). She was admitted on 8 December 2002. On admission, she was well-orientated (blood pressure: 110/65 mmHg; heart rate: 70 beats/min). ADH levels were 0.5 ng/l and she had low urine osmolality (98 mOsm/kg) and high plasma osmolality (307 mOsm/kg). Her serum Na was 144 mmol/l, and serum K was 4.2 mmol/l. Her serum total triiodothyronine was 2.15 nmol/l (reference range 0.91–2.70 nmol/l), serum total thyroxine was 106 nmol/l (reference range 58–115 nmol/l), and serum thyrotropin was 1.5 mU/l (reference range 0.3–4.0 mU/l). She had negative thyroglobulin-, thyroid peroxidase-, and pituitary-antibodies. Her morning plasma cortisol level was 405 nmol/l (reference range 160–810 nmol/l), and adrenocorticotropin was 7.15 pmol/l (reference range less than 13.2 pmol/l).

The diagnosis of CDI was made on the basis of the decreased ADH levels with low urine osmolality and high plasma osmolality. T1-weighted MRI (Fig. 1A,D) revealed prominent pituitary-stalk thickening and neurohypophysial enlargement, indicating lymphocytic infundibuloneurohypophysitis that caused CDI. Her CDI was controlled with vasopressin. She was not treated with steroids. She recovered from CDI spontaneously and vasopressin was discontinued.

T1-weighted MRI (Fig. 1A,D) revealed prominent pituitary-stalk thickening and neurohypophysial enlargement. An MRI scan in March 2003 (Fig. 1B,E) demonstrated improvement of pituitary-stalk thickening and neurohypophysial enlargement. An MRI scan in June 2004 (Fig. 1C,F) revealed an empty sella. With improvement of pituitary-stalk thickening and neurohypophysial enlargement, she recovered from CDI. The serial MRI changes – prominent pituitary-stalk thickening and neurohypophysial enlargement (A, D), their improvement (B, E), and then the empty sella (C, F), were associated with the clinical course of the disease. She was not treated with steroids. The serial MRI changes might show a natural course of lymphocytic infundibuloneurohypophysitis.

Figure 1 Serial T1-weighted magnetic resonance imaging (MRI) of a patient with central diabetes insipidus (CDI) possibly due to lymphocytic infundibuloneurohypophysitis. T1-weighted MRI in December 2002 (A, D) revealed prominent pituitary-stalk thickening and neurohypophyseal enlargement, indicating lymphocytic infundibuloneurohypophysitis, which caused CDI. MRI in March 2003 (B, E) demonstrated improvement in the pituitary-stalk thickening and neurohypophyseal enlargement. MRI in June 2004 (C, F) revealed empty sella. A, B, C: coronal view; D, E, F: sagittal view.

In our patient, the initial MRI findings were prominent pituitary-stalk thickening and neurohypophyseal enlargement, suggesting that she had lymphocytic infundibuloneurohypophysitis (3–5). Lymphocytic infundibuloneurohypophysitis is typically observed in women during late pregnancy or in the postpartum period but may be seen at any age and in either gender. We demonstrated the serial MRI changes in a patient with CDI possibly due to lymphocytic infundibuloneurohypophysitis. The MRI changes are associated with the clinical course. Steroids were not administered to the patient and she recovered from CDI.

We demonstrated serial T1-weighted MRI changes in a patient with lymphocytic infundibuloneurohypophysitis. CDI can be caused by lymphocytic infundibuloneurohypophysitis, which can be detected by MRI. The clinical course of the disorder is associated with T1-weighted MRI changes.

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