of a postnatal neurologic injury, no compensatory rise in TSH levels, and normal thyroid structure without thyroid autoantibodies, raised a suspicion of CH rather than primary hypothyroidism. Therefore, a combined pituitary function stimulation test with an intravenous injection of regular insulin (0.15 U/kg), TRH (400 μg), and luteinizing-hormone-releasing hormone (LHRH; 100 μg) was performed (Table 1). This showed a normal rise in TSH, with decreased cortisol and growth hormone (GH) responses. To determine the origin of the hormonal deficiency, 1 g/kg body weight of corticotropin-releasing hormone (CRH) was administered; the adrenocorticotropic hormone (ACTH) response was normal, suggesting normal pituitary function and a possible hypothalamic defect (Table 2).

DISCUSSION

Dyke et al 8 first described DDMS in 1933 after observing the radiological features of nine patients with cerebral hemiatrophy combined with ipsilateral ventricular dilatation, compensatory osseous hyper trophy, and hyperpneumatization of the ipsilateral mastoid cells and paranasal sinuses. 7 The clinical features of DDMS vary and include seizures, contralateral hemiparesis, facial asymmetry, and mental retardation according to the extent of brain injury. 6 DDMS may be congenital or acquired from various etiologies. 3,5,9 The intrauterine cause of the congenital form is generally unknown; however, cases with vascular abnormalities 10 and coarctation of the midaortic arch 11 have been reported, suggesting involvement of vascular factors in the brain damage. The causes of acquired DDMS are variable and include infection, trauma, ischemia, and hemorrhage. 12 We believed our patient had acquired DDMS because she developed neurological abnormalities after severe meningitis at 12 months of age. Furthermore, the radiological findings supported an acquired cause of the disease. The presence of prominent sulci suggested that the brain insult occurred after development of the cerebral parenchyma rather than during embryogenesis. 4 Compensatory skull thickening and enlarged mastoid cells indicated that the brain insult had occurred before the patient was 3 years of age and prior to maturation of the calvarium. 3 The patient developed late seizures at 7 years of age. We are unable to explain the time lag between meningitis at 12 months of age and the seizures at age 7; however, it may be related to poor development of the cerebral hemisphere causing a decrease in neural activity and synaptic plasticity as a result of low neurotrophin levels. 13

Hypothyroidism refers to thyroid hormone deficiency that usually stems from primary thyroid failure caused by chronic autoimmune thyroiditis and, very rarely, from central dysfunction of the pituitary or hypothalamus. 14 Although a TSH measurement is the preferred screening test for thyroid insufficiency,