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Case-control studies investigating the different types of neurologic deficits associated with classic Gaucher's disease and type 1 Gaucher's disease are reviewed ([@B1]). The pattern of neurological involvement includes spastic paraparesis, cerebellar ataxia, dementia, and chorea. The severity of neurological involvement is directly correlated with the duration of acid-β-glucosidase deficiency. Although the majority of patients with clinically diagnosed type 1 Gaucher's disease are diagnosed with the more severe manifestation (neuronopathic type 1 Gaucher's disease), some patients with the non-neuronopathic type 1 Gaucher's disease have some degree of neurologic involvement ([@B4]). In this review, the authors demonstrate an interesting case of a patient who was ultimately diagnosed with non-neuronopathic type 1 Gaucher's disease and later developed a partial disease at his first MRI examination 4 years after diagnosis. Although imaging studies are typically performed after the diagnosis of the neurological manifestations of Gaucher's disease, it should be emphasized that such techniques are not only useful to confirm the diagnosis of neurological disease in patients with the disease. This case is highly relevant as the patient was erroneously diagnosed with classic Gaucher's disease based on the typical presentation of the disease. After receiving the diagnosis and treatment for classic Gaucher's disease, the patient developed some liver dysfunction at first and later developed a neurological manifestations of the disease. Several other studies have reported an increased risk of glucocerebrosidase deficiency among family members with the disease ([@B2],[@B5]). Based on these observations, it is necessary to perform molecular analysis of the *GBA1* gene if a patient has a typical clinical presentation of the disease. This is particularly true in cases with an atypical presentation, such as the patient discussed herein who initially presented with only classical neurologic manifestations and later developed a partial disease. We also reviewed the literature on the association between *GBA1* mutations and spinocerebellar ataxia type 2 (SCA2). This type of ataxia is considered to be closely related to SCA3, a subtype of autosomal dominant cerebellar ataxia, although the molecular mechanisms responsible for these diseases remain unclear ([@B6]). More than 10 mutations in the *SCA2* gene have been reported, including four that cause the protein truncation or missense mutations ([@B7]). Although it is difficult to evaluate the frequency

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