[within the first year of life] was 5.1±1.6, as compared with 6.8±2.1 for subjects with congenital leptin deficiency (P=0.005) and 5.0±1.5 for MC4R-deficient subjects. Adults with *LEPR* had hypogonadotropic hypogonadism. These probands consumed almost three times the amount of energy as control subjects. Circulating leptin levels were not disproportionately elevated in these subjects, suggesting that serum leptin couldn't be used as a marker for leptin-receptor deficiency.[19] *LEPR* deficiency should be evaluated in all patients with severe obesity in the absence of developmental delay and dysmorphic features. This is important in genetic counselling of patients, and deciding the treatment regimen.

***SDCCAG8*:** An additional variant c.1513C>G, p.(Gln505Glu) was identified in the *SDCCAG8* gene. This was classified as a VUS. When a VUS is reported, we do not recommend use of genetic information in risk stratification, patient management or genetic counseling.

Data from the "Guidelines (2013) for the Management of Overweight and Obesity in Adults" recommended the following lifestyle interventions to achieve and maintain a 5-to-10% reduction in body weight: counseling, diet, physical activity, and behavioral therapy.[3,20] Obesity management is multifactorial with emphasis on long-term vigilance, psychological support, and weight management.[20,21] The patient was discharged after bariatric surgery. Her weight at discharge was 262 kilograms.

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