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SPONTANEOUS HASHIMOTO'S THYROIDITIS REMISSION IN TWO CONSECUTIVE PREGNANCIES

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INTRODUCTION

Hashimoto's thyroiditis (HT), a type of autoimmune thyroid disorder, is the most common cause of hypothyroidism. The course of HT is altered during pregnancy with majority of cases requiring an increment in levothyroxine doses as high as 20-40% to achieve euthyroidism. Spontaneous remission of HT during pregnancy is extremely rare.

CASE

We describe a 37-year-old female who was diagnosed with Hashimoto's thyroiditis initially presenting with neck swelling and symptoms of hypothyroidism seven months after her first pregnancy. Her thyroid function test showed overt hypothyroidism (TSH 83.9 IU/ml, FT4 3.9 pmol/L) with positive anti-thyroid peroxidase antibody (TPO Ab) at 196 IU/ml (>34), and thyroid stimulating immunoglobulin of less than 0.1 IU/L. She was started on levothyroxine 100 mcg daily. Patient had a miscarriage during her second pregnancy. On her third pregnancy, she developed hyperthyroidism at 10 weeks of gestation, requiring gradual reduction of her thyroxine and eventual discontinuation at 2 months postpartum. Thyroxine was resumed 6 months postpartum due to overt hypothyroidism. A similar pattern was observed in her fourth pregnancy wherein she developed hyperthyroidism at 13 weeks also requiring gradual reduction and later discontinuation of thyroxine at 35 weeks of gestation. Anti-TPO Ab at 35 weeks of gestation was still elevated at 158.2 IU/ml. Her pregnancy was uneventful and she delivered a healthy baby. She remains in remission three months postpartum.

CONCLUSION

Spontaneous remission of HT can occur during pregnancy, usually beyond the second trimester., However, recurrence of HT has been observed during the postpartum period. Thyroid autoantibodies may play a role in these changes. Close monitoring of thyroid function is essential.

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RARE PRESENTATION OF A GIANT INTERNAL CAROTID ARTERY (ICA) ANEURYSM CAUSING HYPOPITUITARISM

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INTRODUCTION

Pituitary insufficiency secondary to giant internal carotid artery (ICA) aneurysms are rare. It is, however, an important differential to consider in cases of hypopituitarism as prompt diagnosis and treatment is necessary to prevent a fatal outcome.

CASE

We present a case of hypopituitarism with hyperprolactinemia secondary to a suprasellar giant ICA aneurysm. A 60-year-old female presented with bilateral visual field defects. Examination showed left temporal hemianopia and right inferior scotoma. Her cranial CT revealed a well-defined, rounded lesion arising from the suprasellar region. She was then referred for a brain and pituitary MRI. While awaiting the date of her MRI appointment, she developed diarrhoea and increasing lethargy.

Pituitary hormone panel done demonstrated panhypopituitarism with hyperprolactinemia: cortisol 12 nmol/L (normal range 185-624), TSH 1.238 mU/L (0.34-5.6) free T4 6.8 pmol/L (7.9-4.4), FSH 2.2 mU/ml (2.5-10.2), LH 0.2 mU/ml (15.9-54), prolactin 1400 u/ml (<500) and GH 0.11 Ug/L (0.077-5.0). The hyperprolactinemia was attributed to the stalk compression effect. She was given hydrocortisone and thyroxine replacement. MRI revealed a sellar and suprasellar mass suggestive of a distal right ICA aneurysm. The findings were confirmed by digital subtraction angiography. Cerebral stenting was successfully performed. One month postoperatively, she has not shown any recovery of pituitary function.

CONCLUSION

We described an unusual case of a suprasellar giant ICA aneurysm leading to pituitary insufficiency. ICA aneurysms, particularly those located at the sellar-suprasellar region, are very rare. However, since they resemble pituitary tumours in terms of imaging and laboratory findings, an accurate diagnosis must be made in order to institute necessary treatment strategies among these patients.