Case report: Combined hypothyroidism and hypoparathyroidism in an infant following maternal administration of Iodine^{131} in early pregnancy

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Introduction

The relationship between maternal Iodine^{131} administration and neonatal hypothyroidism is relatively well recognised [1]. The thyroid gland can already be seen by 7 weeks of gestation and thyroid follicles containing colloid are evident histologically by 10 weeks. T3 and T4 can be detected at 12 weeks. At approximately this time the foetal thyroid gland can accumulate radioactive iodine [1].

Hypoparathyroidism is a known but rare complication of I131 therapy in adults, but not widely reported in neonates [2].

Parathyroid tissue can first be seen at 5 weeks gestation [3]. At approximately 7 weeks, the parathyroid glands migrate from their origin in the third and fourth pharyngeal pouches to their final position on the posterior surface of the thyroid gland. Parathyroid hormone secretion starts at 12-13 weeks. The foetal thyroid gland is able to accumulate radioactive iodine at this time, so it is reasonable to hypothesise that high concentrations of I131 can also damage the small amount of the parathyroid tissue located on the dorsal aspect of the thyroid gland, as β particles from I131 can penetrate tissue to a depth of 2mm

We report the first UK case of hypoparathyroidism secondary to maternal Iodine^{131} therapy

Case presentation

A 27 year old woman received two doses radioactive I131 therapy following total thyroidectomy for thyroid carcinoma. The first dose was given approximately three months prior to conception and the second dose at an estimated 10-12 weeks gestation. The mother was put on thyroid hormone replacement therapy and was on 200 mcg levothyroxine at the end of pregnancy.

A female infant was born at 39 weeks gestation, weighing 3400g with a normal Apgar score. At birth she was found to have features of severe hypothyroidism, with a capillary TSH >150mU/L on day 1 of life, 268 mU/L day 6. She was normocalcaemic on day 1 (2.21mmol/l) but became hypocalcaemic by day 2 (1.43mmol/l,PTH<1.2pmol/l), with normal vitamin D levels. The infant was initially commenced on daily levothyroroxine (25mcg) and calcium alone. Plasma calcium normalised; supplements were reduced then discontinued at day 25 with ongoing monitoring. She became hypocalcaemic again at day 37 and 1 Alpha Calcidol was commenced.

At the age of 6 weeks the patient experienced a cardiorespiratory arrest, secondary to RSV bronchiolotits and hypocalcaemia. Blood results at the time showed: calcium 1.23mmol/L, phosphate 3.55mmol/L, PTH <1.0pmol/L, free T4 12.7pmol/L, TSH 21.00mu/L. Unfortunately, the patient was apnoeic for more than 10 minutes and as a consequence suffers from total body dyskinetic cerebral palsy (secondary to ischaemic encephalopathy). The patient also proceeded to experience daily myoclonic seizures.

Conclusion

Radioactive iodine (RAI) therapy is contraindicated in pregnancy, however, its use in undetected pregnancy has been reported [6]. Our patient was exposed to radioactive I131 early on in the pregnancy. This has led to the development of hypothyroidism and hypoparathyroidism in-utero. Radioactive iodine readily crosses the placenta and foetal serum RAI level can reach 75% of the mother's serum level, persisting in the foetal thyroid gland for 70-75 days [7]. In this case report the administration of I131 in early pregnancy had resulted in persistent neonatal hypothyroidism with a TSH of >150mU/L at birth.

Maternal administration of I131 in early pregnancy can lead to combined hypothyroidism and hypoparathyroidism in the neonate; this can have devastating consequences for the infant's future development. In this case the severity of this infant's hypothyroidism also questions maternal compliance with her own thyroxine medication Effective safety and screening measures should be undertaken in all women of childbearing age to exclude pregnancy prior to as well as during radioactive iodine therapy. If the foetus has been exposed to radioactive iodine, foetal thyroid and parathyroid function should be monitored to assess the risk of developing complications.

Summary

Hypoparathyroidism, although rare, has been reported and is a recognised complication of radioactive Iodine therapy in adults. Hypothyroidism has been reported in neonates who have been exposed to Iodine^{131} in-utero, however, only one case of neonatal hypoparathyroidism secondary to maternal Iodine^{131} therapy has been described in the literature. To our knowledge this is the first case in the UK.

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