FOLLOW-UPS OF CONGENITAL HYPOTHYROIDISM

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ABSTRACT

Brain development depends on thyroid hormones, the delay in starting treatment of hypothyroidism leads to a permanent mental impairment which will be more severe, the longer the delay of the treatment. So neonatal screening for congenital hypothyroidism is very important, as is being done in other countries. Congenital hypothyroidism is common in northern Bangladesh, but can be cured, if treated early as documented by the six cases presented hear.

Key words : Congenital hypothyroidism

MATERIALS AND METHODS

Among about 6000 patients at the Institute of Nuclear Medicine Dhaka, Nuclear Medicine centres at Dinajpur & Rangpur, examined by various radioisotope techniques e.g. Radioiodine Uptake (RAIU), Thyroid scan, RIA of T3, T4, & TSH. We looked for congenital hypothyroidism.

RESULTS : Results are presented case-wise.

CASE 1

A girl, 4 years in chronological age, but 2 years in bone age (2 carpal bones present), with dull look came at N.M.C, Dinajpur in 1991. Her mother complained that the baby was constipated, her appetite was poor and her growth was retarded both physically and mentally---she could not walk and talk. Her radioiodine uptake and serum levels of thyroid hormones were low (RAIU 2 h. 1.88%, 24 h. 0.34%), (Table-1). She was given thyroxine tablets at first 12.5 micrograms/day and gradually the dose was increased to 25 mcg/d, then 50 mcg/d in 1995. Since then she was being followed up at N.M.C, Rangpur --- she was of quite normal look, her memory became good, bone age was 7 years in 1995, her school performance was also quite satisfactory (stood 9th among 45 students).

She was taking 75 mcg/day thyroxine (occasionally irregular) since 1998 and experienced menarche at 12 years of age in Apr. 1999. Her secondary sex characters were also normal. She was advised to increase thyroxine to 100 mcg daily on 25.10.99, due to high TSH level.

CASE 2

Daughter of a physician started thyroxine therapy at 5 mo. of age, had irregular medication due to nonavailability, however, physically & mentally well, started menarche at 13y. 6m. (Sep. 1999). In Sep. 1998 her T₃ was 1.7 nmol/L, T₄ was 188 nmol/L, TSH was 56.5 mlU/L and increased the dose of thyroxine to 125 mcg/day.

CASE 3

Another girl of 18 yrs. age came here for

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thyroid investigations with a history of thyroxine therapy for last 14 yrs. for congenital hypothyroidism she was taking 250 micrograms of thyroxine daily, married on 5.12.99, but having occasional polymenorrhea and constipation probably due to irregular medication. On 19.12.99 her T_3 was 1.8 nmol/L, T4 was 375 nmol/L, TSH was 0.5 mlU/L and she was advised to reduce the dose of thyroxine to 150 mcg/day.

CASE 4

A girl of age 12 yrs. treated by Carbamazepine and clobazam for epilepsy and dull memory, had $T_3 = 1.01 \text{ nmol/L}$, $T_4 = 18 \text{ nmol/}$ L and TSH = 115 mlU/L on 27.11.96 and started thyroxine therapy (100 mcg/day), she improved her intelligence and had menarche in 1997 and increased the dose of thyroxine to 150 mcg/day.

Table 1. Hormone levels of case 1

On 23.12.98 her T_3 was 1.9 nmol/L, T_4 was 42 nmol/L and TSH was 2.5 mlU/L.

CASE 5

A girl of age 6 months presented with constipation of one week in June 1999. Her T_3 was 0.35 nmol/L, T_4 was 9 nmol/L, and TSH was 87.5 mlU/L. She was improved by thyroxine (12.5 mcg/day), as confirmed by follow-ups done at 3 and 6 months of intervals.

CASE 6

A female baby of age 4 months presented with oliguria and constipation on 4.1.2000. Her T_3 was 0.15 nmol/L, T_4 was 7 nmol/L, and TSH was 100 mlU/L. She was improved by thyroxine tablets 12.5 mcg/day.

Date	T ₃ NR 0.8-3.16 nmol/L	T ₄ NR 64.5-152 nmol/L	TSH NR 0.3-6 mlu/L
17. 2.91	0.94	<20	
13. 8.98	2	115	95
25.10.99	1.7	114	100

NR = Normal range.

DISCUSSION

In 1908, McCarrison first described the two forms of cretinism- hypothyroid and neurologic.¹ Incidence of congenital hypothyroid was 1 in every 3000 to 6000 live births in Europe (1979)² and America (1976)³ In iodine-sufficient populations about 1 in 4000 neonates has congenital hypothyroidism usually from inadequate thyroid development,⁴ screening program was advised⁵ but not yet implemented fully.⁶ Dose of thyroxine is to be monitored regularly-it should always be taken as a single daily dose as it has a plasma half-life of about seven days. The correct dose of thyroxine is that which restores serum TSH to normal. Hormonal assays should be done at least as often as 4 weeks after each change in thyroxine dosage as well as 2 and 6 wks. after initiation of therapy and at 3, 6, 9, 12 and 18 months of age and on each subsequent birthday.⁷ Neonatal screening program in Poland shows that some increased TSH levels (9% versus 3.2%) are from the use of antiseptic iodine and this must be remembered in using the neonatal TSH in screening for iodine deficiency.⁸ Fetal hypothyroidism can occur secondary to treatment of maternal hyperthyroidism or due to congenital thyroid defects and may be cured both by intraamniotic levothyroxine(LT_4) injection 250 or 500 mcg/wk. or maternal administration of the same drug. The transplacental transfer of L-T₄ is demonstrated by the level of free T₃ in fetal blood.^{9,10}

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