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Hypothyroxinemia And Strabismus (Squint)

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ABSTRACT

Hypothyroxinemia and strabismus is a serious problem for Bangladesh as well as in the world. For this reason blindness occur in the world.It is an avoidable cause of blindness and has a global prevalence, which ranges from 2% to 6%. Low level of thyroid hormone thyroxin called hypothyroxinaemia is responsible for it. In different studies and analysis, the role of thyroid hormones at different phases of brain and neural development and maturation occurs and it is important and draws our attention for the vulnerable periods. These periods occur during gestation and lactation when genetic (nature) environmental (nurture) factors, which include nutrients and chemical contaminants, interfere with maternal and offspring thyroid health. Iodine deficiency from inadequate alimentary habits is the most common cause of maternal and foetal thyroid dysfunction. In addition, selenium (a component of deiodinases), iron (a component of the prosthetic heme group associated to thyroperoxidase) and other micronutrients are required for an adequate life-long thyroid function, especially during development and adolescence.Moreover, environmental anti-thyroid contaminants are acquiring increased importance for interference of thyroid hormone synthesis, transport and metabolism. Besides these, some auto-immune thyroid diseases also interfere thyroid hormone metabolism and help to develop the complications of eyes.

Key words: Hypothyroxinemia, blindness, strabismus, thyroid hormones, eyes-investigative parameters, iodine deficiency disorders, binocular vision, diplopia, rivalry, disparity, teratogeniceffects, pleiotropic action, genetic mutation, cretins, nystagmus, diplegia, confusion

INTRODUCTION

Binocular single vision (BSV) is one of the hallmarks of human race and it is the best criteria on the supremacy gained by the animal kingdom. It is not without reason that about 60% of the brain tissue and more than half of the twelve cranial nerves sub-serve the functions of eyes. BSV is accomplished by a perfect sensor motor coordination of the two eyes, both at rest and during movement. The two two-dimensional images of an object of interest formed at the fovea of each eye, transmitted to the respective visual cortex and processed and perceived as one three-dimensional (3-D) percept. This requires constant and controlled activity of the appropriate eye muscles to maintain fixation of the two eye-cameras on the concerned object, irrespective of the movement between it and the observer. It also requires the accommodation mechanism to maintain clear view even as the object moves closure or further.

Normally, the two visual axes meet at the point of regard or the object of attention. The eyes are said to be in alignment (orthophoriaor orthotrophia) due to some unfortunate reason the two visual axes may not aligned to the point of regard, that is one eye fixates at the point but the other eye does not. A strabismus or squint results.

It is an avoidable cause of blindness and has a global prevalence, which ranges from 2% to 6%. Low level of thyroid hormone thyroxin called hypothyroxinaemia is responsible for it. In different studies and analysis, the role of thyroid hormones at different phases of brain and neural development and maturation occurs and it is important and draws our attention for the vulnerable periods. These periods occur during gestation and

lactation when genetic (nature) environmental (nurture) factors, which include nutrients and chemical contaminants, interfere with maternal and offspring thyroid health. Iodine deficiency from inadequate alimentary habits is the most common cause of maternal and foetal thyroid dysfunction. In addition, selenium (a component of deiodinases), iron (a component of the prosthetic heme group associated to thyroperoxidase) and other micronutrients are required for an adequate life-long thyroid function, especially during development and adolescence.

Moreover, environmental anti-thyroid contaminants are acquiring increased importance for interference of thyroid hormone synthesis, transport and metabolism. Besides these, some auto-immune thyroid diseases also interfere thyroid hormone metabolism and help to develop the complications of eyes.

Examination of the eyes, and other investigative parameters, such as duration of iodine deficiency in a locality, thyroid hormone bioassay may be an indicator to assess its viability.

My study is to find out this relation of hypothyroxinemia with strabismus development along with neurological form of cretinism which are more commonly found along with it. So, at the early stages, if the precautions and preventive measures are taken, then miseries and burden can be reduced, and so surgical or curative intervention is not required thereafter.

OBJECTIVESOF THE STUDY

The objectives of the study are as follows:

1. To find out the prevalence of strabismus with hypothyroxinaemia in Bangladesh.
2. To evaluate the status of IDD in Bangladesh.
3. To assess the findings and relationship between the autoimmune thyroid diseases (thyroiditis) and hypothyroxinaemia.
4. To provide a guideline in reducing amblyopia.

MATERIALS AND METHODS

Study Area: The study was carried out mostly in endemic prone areas, like- Jaldhakaupazila and around to it in Nilphamari district, Bangladesh.

Study Design: It was a descriptive and investigative cross-sectional institute based study

Sources of Data: Data were collected from primary and secondary sources.

Sources of Primary Data: Primary data were collected from the respondents of the study areas.

Sources of Secondary Data: Secondary data were collected from books, research reports, journals, different websites and internet etc.

Sampling Method: Purposive and convenience sampling was followed for data collection.

Sample Size: Total 300 respondents were the sample size.

Tools for Data Collection: Questionnaire was used for data collection

Method of Data Collection: Primary data were collected by face to face interview, clinical examination, specimen sample collection and secondary data were collected by reviewing secondary sources of data.

Data Analysis: Data were analyzed by using by using suitable computer program- like MS Word, Excel and SPSS.

Presentation of Findings: Findings were interpreted and presented in the thesis.

Pre requisite- equipments and appliances

The examination of strabismus requires some essential equipments, those include:

1. Torch light,
2. Occluder,
3. Fixation targets : for distance and near to control the accommodation as desired,
4. RAF rular,
5. Trial set with prisms of 1 8 Pd ,
6. Bagolini'sstreated glasses,
7. Red and green goggles,
8. Double Maddox rod set,
9. Snellen's distant vision chart with letters and E in rows, a single letter E chart and Near vision chart,
10. A Protractor with a foot rular,
11. Ophthalmoscope (direct).

RESULTS

Table 1: Age of the respondents

Age	Frequency	Percent
10-20 years	220	73.3
21-30 years	42	14.0
31-40 years	38	12.7
Total	300	100.0

Table 2: Gender of the respondents

Gender	Frequency	Percent
Male	142	47.3
Female	158	52.7
Total	300	100.0

Table 3: Literacy of the respondents

Gender	Frequency	Percent
Male	142	38.9
Female	158	61.1
Total	300	100.0

Table 4: Religion of the respondents

Name of Religion	Frequency	Percent
Islam	250	83.3
Hindu	50	16.7
Total	300	100.0

Table 5: Occupation of the respondents

Name of occupation	Frequency	Percent
Farmer	124	41.3
Labour	63	21.0
Business	25	8.3
Mechanic	25	8.3
Government service	38	12.7
Private service	25	8.3
Total	300	100.0

Table 6: Income of the respondents

Income	Frequency	Percent
2000	25	8.3
15000	37	12.3
20000	76	25.3
40000	112	37.3
50000	25	8.3
60000	25	8.3
Total	300	100.0

Table 7: Dietary habit of the respondents

Dietary Pattern	Frequency	Percent
Vegetarian	50	16.7
Non-vegetarian	250	83.3
Total	300	100.0

Table 8: Consumption of sea fish or sea vegetables

	Frequency	Percent
Yes	110	36.7
No	190	63.3
Total	300	100.0

Table 9: Reason of eating sea fish or sea vegetables

	Frequency	Percent
Tasty	62	20.7
Rich in iodine	50	16.6
Available	25	8.3
Cheap	38	12.7
Good for health	125	41.7
Total	300	100.0

Table 10: Reason of not eating sea fish or sea vegetables

Reason of not eating	Frequency	Percent
Costly	190	63.3
Total	190	100.0

Table 11: Frequency of eating sea fish or sea vegetables

	Frequency	Percent
Twice weekly	149	49.7
Thrice weekly	63	21.0
Once in a month	63	21.0
Total	275	91.7

Table 12: Cooking food with iodized salt

	Frequency	Percent
Yes	300	100.0

Table 13: Reason of cooking food with iodized salt

	Frequency	Percent
Self interest	300	100.0

Table 14: Have power disturbance or refractive errors of family members

	Frequency	Percent
Yes	50	16.7
No	250	83.3
Total	300	100.0

Table 15: If yes the reason of

		Frequency	Percent
Valid	Mother	50	16.7
Missing	System	250	83.3
Total		300	100.0

Table 16: Have any deviations or changes of deviations of direction of eyeballs

		Frequency	Percent
Valid	Yes	300	100.0

Table 17: Directions of deviations

Directions of deviations	Frequency	Percent
Inwards	150	50.0
Outwards	150	50.0
Total	300	100.0

Table 18: Frequencies of changes occur

Changes occur	Frequency	Percent
Sudden	113	37.7
Gradual	187	62.3
Total	300	100.0

Table 19: Seen by whom

	Frequency	Percent
By yourself	62	20.7
By others	238	79.3
Total	300	100.0

Table 20: Close relatives suffered from such deviation of eye conditions

	Frequency	Percent
Yes	25	8.3
No	238	79.3
Total	263	87.7

Table 21: If yes name of close relative

	Frequency	Percent
Mother	25	100
Total	25	100

Table 22: Any Close relatives suffered from visible enlarged thyroid gland

	Frequency	Percent
Yes	88	29.3
No	212	70.7
Total	300	100.0

Table 23: If yes who suffered from visible enlarged thyroid gland

	Frequency	Percent
Mother	88	100
Total	88	100

Table 24: Have any sign or symptoms of enlarged thyroid gland in the past or present

	Frequency	Percent
No	300	100.0

Table 25: Done any investigational procedure, like thyroid hormone bioassay of blood or urinary iodine estimation

	Frequency	Percent
Yes	62	20.7
No	238	79.3
Total	300	100.0

Table 26: Reason of investigation procedure like thyroid hormone bioassay of blood or urinary iodine estimation

	Frequency	Percent
Advice of doctor	62	100
Total	62	100

Table 27: Reason of not investigation procedure like thyroid hormone bioassay of blood or urinary iodine estimation

	Frequency	Percent
Not interested	38	12.7
Fear	25	8.3
For complex interaction	63	21.0
To become a patient	112	37.3
Total	238	79.3

Table 28: Have suffered any hearing loss or articulation of speech or any member of family suffer from

	Frequency	Percent
No	300	100.0
Total	300	100.0

Table 29: Have any weakness spasticity, rigidity or stiffness of muscles of limbs

	Frequency	Percent
No	297	99.0
Present	3	1.0
Total	300	100.0

Table 30: Visual Acuity determination in right eye

	Frequency	Percent
Normal	50	16.7
Abnormal	213	71.0
Total	263	87.7

Table 31: Visual Acuity Distance vision left eye

	Frequency	Percent
Normal	175	58.3
Abnormal	88	29.3
Total	263	87.7

Table 32: Visual Acuity Distance vision pinhole in right eye

	Frequency	Percent
Improved	63	21.0
Not improved	175	58.3
Total	238	79.3

Table 33: Visual Acuity Distance vision pinhole in left eye

	Frequency	Percent
Improved	138	46.0
Not improved	25	8.3
Total	163	54.3

Table 34: Near vision in both eye

	Frequency	Percent
Normal	225	75.0
Abnormal	38	12.7
Total	263	87.7

Table 35: Corneal light reflex test (H.R.)

	Frequency	Percent
Esodeviation Alt	70	23.3
Exodeviation	80	26.7
Cemtrally placed	150	50.0
Total	300	100.0

Table 36: Cover test

Cover test	Percentage
Exophoria in right eye	7.69%
Exophoria in left eye	0%
Esophoria in right eye	3.84%
Esophoria in left eye	0%
Alternate exophoria	11.53%
Alternate esophoria	15.38%
Exotrophia right eye	3.84%
Exotrophia left eye	0%
Esotrophia right eye	7.69%
Esotrophia left eye	0%
Alternate exotropia	0%

Alternate esotropia	0%
Normal	53.85%

Table 37: Ocular motility test

Ocular motility	Percentage
Normal	80.77%
Restricted	15.38%
Nystigmus	3.84%

Table 38: Fundus/ ophthalmological examination

Fundus examination	Percentage
Normal	96.15%
Abnormal	0%
Not visualized	3.85%

Table 39: Urine for urinary iodine

µg/L	Frequency	Percent
1.06	25	8.3
7.2	25	8.3
8	25	8.3
9.29	25	8.3
9.78	25	8.3
11.46	25	8.3
12.9	38	12.7
19.99	37	12.3
23.58	38	12.7
35.7	37	12.3
Total	300	100.0

Table 40: Blood test for serum Thyroglobulin

ng/ml	Frequency	Percent
0.55	37	12.3
1.806	25	8.3
2.08	25	8.3
7.1	37	12.3
9.86	38	12.7
11.81	25	8.3
13.11	25	8.3
16.89	25	8.3
34.26	25	8.3
60.61	38	12.7
Total	300	100.0

Table 41: Blood test for serum T.S.H.

mlu/ml	Frequency	Percent
1.84	25	8.3
1.92	25	8.3
2.38	37	12.3
2.41	63	21.0
2.54	25	8.3
2.64	25	8.3
2.67	37	12.3
3.43	25	8.3
5.65	38	12.7
Total	300	100.0

Table 42: Blood test for serum FT4

pmol/L	Frequency	Percent
12.52	38	12.7
15.64	50	16.7
16.38	25	8.3
17.02	38	12.7
17.25	62	20.7
18.3	25	8.3
20.25	37	12.3
21.16	25	8.3
Total	300	100.0

Table 43: Blood test for serum FT3

pmol/L	Frequency	Percent
6.2	38	12.7
6.81	37	12.3
7.48	25	8.3
7.69	62	20.7
8.27	38	12.7
8.58	25	8.3
9.1	25	8.3
10.5	25	8.3
11.51	25	8.3
Total	300	100.0

DISCUSSION

Eyes are the sensory organs of vision. The visual perception is received by the sensory stimulus and interpreates it through the brain, visual cortex. There are some extra ocular and intraocular muscles which act through cranial nerves connection to the brain by which an object in the form, position, color and contrast discrimination is perceived. The two eyes acts as a camera through which binocular vision perceived. In the visualizing process, there is also some advantages which are, single vision, fusion, and its depth perception. To see and observe any object in details, both eyes are move in that direction, or along the movement of an object in that direction. The eyes are move in their axis of movement in different directions and close or distant movements with the help of different groups of ocular muscles and cranial nerves.

There is also accommodation and convergence of eyes occur when this types of movement is required. There is a certain critical period of time when the development commences. During this development process some essential allosteric and metabolic factors are required with their co-ordinating action. Thyroid hormone, thyroxin (T4) is such an essential metabolite which is required proper organic development in that time. The specific consequences of T4 deficiency depends on the precise developmental timing of the deficiency. If the deficiency occurs early in pregnancy, offspring display problems in visual attention, visual processing (i.e. acuity of vision and strabismus) and gross motor skills.If it occurs later in pregnancy, children are at additional risk of subnormal visual attention (i.e. contrast sensitivity) and visuospatial skills development as well as slower response speeds and fine motor deficits.

Finally, if thyroid hormone (TH) insufficiency occurs after birth language and memory skills are most predominantly affected.The clinical neurological deficits primarily implicate impairments of the formation of ocular cranial nerves and faulty production of the neuro transmitter of the ocular cranial nerve fibers.

The major motor disorder in neurological cretinism is extrapyramidal lesion, characterized by rigidity and spasticity of muscles. Different studies show that molecular mechanisms of TH action influenced by teratogenic effects of some drugs for these pleitropic actions. At differing time points, TH have particular effects on brain maturation, regulation of neuronal development and microglial proliferation, dendritic arborisation, synaptogenesis, cell migration and myelination.

In this adversely affected brain maturation processes, that potentially result in structural and metabolic brain abnormality visible and evidenced by MRI or MRS and nuclear imaging techniques. So fully developed binocular vision (all grades) of the child requires proper TH effect. Any deficiencies have got in the mother's womb or after delivery of the child, have to develop the visual problems either in motor anomaly strabismus or both sensory deprivations with motor anomaly, strabismic amblyopia.

Hypothyroxinaemia is the root cause of these visual anomalies. As the mothers are the sufferers and their children are contracted as victims. Both mother and child have deficiency of the TH. Sometimes child has to develop congenitaly hypothyroidism. The major causes of hypothyroxinaemia are iodine deficiency, effects of goitrogens and auto immune thyroid diseases. Besides these, selenium, zinc, iron, copper and deficiency of vitamin A plays important and vital rolein action and metabolism of thyroid hormone.Iodine deficiency varied from place to place region to region of the country and world. According the to the finding, most iodide found in ocean and iodide ions in the sea water is oxidized to element iodine, after volatilization into the atmosphere , which returned to the soil by rain, completing the cycle. Due to global warming and climate change, this iodine cycling is slow and incomplete in many regions, so soil and ground water is deficient in iodine. Moreover leaching of iodine from soil and water occurs due to deforestation, intense glaciations, river bank erosion in flooding. Animal and crops grown on these areas are deficient of iodine; lack of consumption of iodine enhances its deficiency.

The daily iodine requirements in children of 9-13 years of age are 120 ug, teens of 14-18 years of age and adults are 150 ug and pregnant teens and women require 220 ug. It has been observed that identified different chemical substances and naturally occurring some food substances are recognized as goitrogens. These substances are usually active only if iodine supply is limited and goitrogen are intake ina long duration. Many of them have been tested in vitroor have been shown to possess anti-thyroid effects in vivo. Some goitrogens are chemical groups acting directly on the thyroid gland. Such goitrogens are found in widely used tuber cassava, cyanogenic glycosides or in the form of thiocyanate or isothiocyanate, which inhibiting transport of iodide into thyroid gland. Certain sulpher containing onion volatiles, disulphide or other goitrin acting on intrathyroidal oxidation and organic binding process of iodide or coupling reaction.

Excess iodide or lithium consumptions interfering with proteolysis, dehalogenation or hormone release. Some vegetables of cruciferous family i.e. cauliflower, cabbage, broccoli, turnip or soybean have indirect goitrogenic effects to interrupts the enterohepatic circulation of the thyroid hormones. When iodine intake is limited, the interfering effects of these substances usually cannot be overcome. Thiocyanate may cross the placenta of pregnant mother and affect the thyroid of the foetus. Deficiency of selenium, zinc, iron, copper and vitamin A may also have goitrogenic effect.Selenium deficiency may have profound effects on thyroid hormone metabolism and possibly also on the thyroid gland itself. In this situation the function of type 1

deiodinase (a selenoprotein) is impaired. Selenium deficiency also leads to reduction of selenium containing enzyme glutathione peroxidase. Glutathione peroxides detoxifies free radicle H2O2, which is abundantly present in the thyroid gland.

An Autoimmune thyroid disease (AITD) is a result of complex interaction between genetic and environmental factors. Incidence mostly in women between child bearing age 30 and 50 years. AITD genes is the HLA-DR gene locus as well as no- MHC genes including thyroglobulin (Tg) and thyroid stimulating hormone (TSH) receptor genes. The major environmental triggers are excess iodine, medication of some drugs e.g.; iodine containing drugs, amiodarone, infections e.g.; rubella and hepatitis C virus acts as indirect precipitator, smoking, stress, sex steroids and pregnancy.

Diagnosis is based on clinical features and supported laboratory investigations. Thyroid disorders are prevalent in women of child bearing age and for this reason commonly present as a pre-existing disease in pregnancy or after child birth. Due to an increase in thyroxin binding globulin as increase in placental type 3 deiodinase and placental transfer of maternal thyroxin to the foetus, the demand of thyroid hormone is increased during pregnancy. Foetal thyroxin is wholly obtained from maternal sources in early pregnancy, since the foetal thyroid gland only becomes functional in the second trimester of gestation. As thyroxin is essential for foetal neurodevelopment, it is crucial that maternal delivery of thyroxin to the foetus is ensured early in gestation. In pregnancyiodide losses through the urine and the feto-placental unit contribute to state of relative iodine deficiency. Thus pregnant women requires additional iodide intake.

A daily iodine intake of 250 ug is recommended in pregnancy but this is not always achieved even in iodine sufficient parts of the world.The necessary increase in thyroid hormone production is facilitated by and partly due to high levels of oestrogen and weak thyroidstimulating effect of human chorionic gonadotropin (HCG) that acts like TSH. Thyroxin (T4) levels rise from about 6-12 weeks and peak by mid gestation, reverse changes are seen with TSH. After women give birth 20-40% is permanently hypothyroid.There is a weak linear correlation between thyroid receptor antibodies with the titer of antibodies against Yearsiniaenterocolitica antigens. It can be considered that Y. enterocolitica infections may play a role in aetiology of Graves's disease. Thyroid and intestinal diseases prevalently co-exist. Hashimatoesthyroideitis (HT) and Graves's disease (GD) are most common autoimmune thyroid diseases (AITD) and often co-occur with Celiac disease (CD) and Non celiac wheat sensitivity (NCWS). This can be explained by the damaged intestinal barriers and the following increase of intestinal permeability, allowing antigens to pass more easily and activate the immune system or cross reacts with extra intestinal tissues repeatedly (leaky gut syndrome).

Additionally, the composition of the gut microbiota has an influence on the availability of essential micro nutrient synthesis, selenium and zinc are needed for converting T4 to T3 and vitamin D assists in regulating immune response. These micronutrients are often found to be deficient in AITDs, resulting in malfunctioning of the thyroid. The gut microbiota largely regulates homeostasis as well as the microbiota, which underlines the symbiotic relationship. Deiodinase activity has been found in the intestinal wall and could contribute to total T3 body levels. Another influencing factor of microbiota is its effects on neurotransmitters such as, Dopamine, which can inhibit TSH.Normal binocular single vision requires clear visual axis leading to a reasonably clear vision in both eyes. The ability of the retinocortical elements to function in association with each other to promote the fusion of two slightly dissimilar images i.e. sensory fusion. The precise coordination of the two eyes for all direction of gazes. So that corresponding retino-cortical element are placed in a position to deal with two images i.e. motor fusion.

It is fact that is established the role of cortical competition in binocular visual development. To start with at birth all cortical cells have potential connections with both eyes. If both eyes are functioning equally, the cortical cells are driven by each eye are equal. About 10% cells are driven by right eye alone and a similar number by the left eye, the rest 80% cells are driven binocularly (the central 20% of these equally by both eyes and the rest have a predominance of the eye or the other). If by any chance one of the eyes is not functioning properly, the cortical cells of one eye are stolen or usurped by the other. This process of competition is entirely reversible in the initial period of plasticity. Monocular deprivation produces a radical alteration in the ocular dominance columns in the striate cortex in favour of normal eye. It is believed that the two eyes compete for synaptic contacts invisual cortex.Monocular deviation diprives that eye and imposes a severe handicap in this contest. As a consequence the deprived eye loses many of the connections

already formed at birth. The ocular dominance columns of the deprived eye shrink and those of the favored eye swell. A similar change is observed in the lamina of the lateral geniculate body (LGB).

Although, there is no competition at this level. The deprived of LGB cells are smaller as they are required to sustain a lesser arbor of axons in the layer of visual cortex. The critical period corresponds to the time phase when the wiring is still malleable. It has been clear that, when the strabismus amblyopia is likely to be corrected is not beyond the age of 12 years.There are varieties of sensory adaptation that occurs in response to clinical situations that disrupt binocular vision. The development of a specific type of sensory adaptation depends on when (age of the patients) the sensory anomaly occurred and the severity and type of binocular disruption. Visually immature patients may develop monofixation syndrome, anomalous retinal correspondence (ARC) or large regional suppression. Where as in visually mature patients may develop diplopia, confusion or rivalry.

Amblyopia by definition refers to a potential loss of sight in one or both eyes, caused by abnormal visual development secondary to abnormal visual stimulation in the absence of ophthalmoscopic or other marked objective signs. Amblyopia is not actually a sensory adaptation, may occurs as a consequence of suppression.

A test for fixation behavior has been tested in each eye with strabismus having vision less than 6/6 snellen's. It can be tested with the help of fixation star of the direct ophthalmoscope. Patient was asked to cover one eye and fixed the star with the other eye. Fixation may be centric (normal of the fovea) or eccentric, which may be unsteady. A steady central fixation is a good prognostic sign. An unsteady but central foveal fixation indicates a possibility of good vision with conventional occlusion, while a steady paramacular or eccentric fixation indicates a poor prognosis.

Factors causing the underlying eye deviation, at the beginning of scientific ophthalmology, the first theories were that mechanical or muscular multifunction caused comitant strabismus. Since deviations could be corrected by mechanical means, it seemed reasonable to assume that mechanical factors were responsible for them. These mechanical factors were held responsible for anomalies affecting the action of antagonistic muscles due to disproportions in their structure, length, cross section (mass), and elasticity, or to anomalies of the insertions. Structural anomalies of the orbits and orbital tissues were also taken into consideration.

Domenici and coworkers described alterations both contractile structures and mitochondria, more pronounced at the scleral myotendinous junction than in the actual muscle belly, in patients with infantile esotropia. Corsi and coworkers reported on alterations of extracellular muscle proprioceptors in this patient group.

Landolt emphasized the importance of determining the amplitude of the horizontal excursions, but he and other supporters of the accommodative theory of strabismus believed that displacement of the excursions was secondary, occurred gradually under the influence of convergence and accommodation, and resulted eventually in contractures of the muscles. As anexcessive amount of accommodation required clearing the retinal image at a given point of fixation distance, generates an excessive amount of accommodative convergence. This occurs, for example in the uncorrected hypermetrope. Accordingly, one generally finds an esophoria present in uncorrected hypermetropes and an exophoria in uncorrected myopes; however neither excessive nor deficient convergence impulses in themselves lead toesotropia or exotropia.

The vast majority of people have adequate motor fusion and therefore are not heterotropic; but if the fusional amplitudes are inadequate or if the fusion mechanism is impaired by some sensory obstacle, the eyes may deviate. Once fusion has broken down, all other etiologic factors (mechanical and innervational as well as accommodative) gain free rein and heterotropia is established. At first it may be intermittent, but in time it will become constant. Refractive errors, through their effect on accommodation, are without doubt one of the prime causes of misalignment of the eyes. Removal of the deviation by corrective lenses in one third of the patients with comitantesotropia is simple and direct evidence for this.

Innervational (Neurologic) causes have been implicated in the etiology and pathologenesis of strabismus. Donders's theory suggests that a specific innervational mechanism exists for esotropia. Paresis of an extraocular muscle may lead to paralytic strabismus. With diminution of the paresis, the paralytic strabismus

tends to acquire characteristics of a commitant strabismus, and this-is known as spread of commitance. A vertical deviation also may reasonably be assumed to be the immediate cause of a horizontal strabismus. A paresis or paralysis of a vertically acting muscle is a gross obstacle to fusion, one almost invariably finds that in an adult with acquired paralysis of such a muscle a horizontal deviation is also present because the pre existingheterophoria has become manifest.

CONCLUSION

As neuropsychological tools have become more sensitive, it has become apparent that even mild thyroid hormone (TH) insufficiency in humane can produce measurable deficits in very neurological functions, and that the specific consequences of TH deficiency depends on the precise developmental timing of the deficiency. If the TH deficiency occurs early in pregnancy, the offspring display problems in visual attention, visual processing (i.e. visual acuity and strabismus) and gross motor skills. If it occurs later in pregnancy, children are at additional risk of subnormal visual (i.e. contrast sensitivity) and visuospatial skills, as well as slower response speeds and fine motor deficits. Finally, if TH insufficiency occurs after birth, language and memory skills are most predominantly affected. Recent studies confirm that the specific action of TH on brain development depends upon developmental timing and studies informing us about molecular mechanism of TH action.

Endemic cretinism presents a coherent clinical picture through varying in severity and emphasis. The clinical neurological deficits primarily implicate impairment of cerebral cortex, cochlea and basal ganglia. The motor disorder in neurological cretinism is extrapyramidal and is characterized by rigidity with the added measure of spasticity. It is consistent with a putaminopallidal lesion, in addition to other elements. The critical effect on brain occurs during the second trimester of intrauterine life. It seems likely that the irreversible effect on brain results from impairment of neurone production.

Thyroid disorders are prevalent in women of child bearing age and for this reason commonly present as a pre-existing disease in pregnancy or after child birth. Due to an increase in thyroxin binding globulin as increase in placental type 3 deiodinase and placental transfer of maternal thyroxin to the foetus, the demand of thyroid hormone is increased during pregnancy. Foetal thyroxin is wholly obtained from maternal sources in early pregnancy, since the foetal thyroid gland only becomes functional in the second trimester of gestation. In pregnancy, iodide losses through the urine and feto-plcental unit contribute to state of relative iodine deficiency. The pregnant women requires additional iodine intake. A daily iodine intake of 250 ug is recommended in pregnancy but this is not always achieved even in iodine sufficient parts of the world.

The necessary increase in thyroid hormone production is facilitated by and partly due to high levels of oestrogen and weak thyroid thyroid stimulating effect of human chorionic gonadotropin (HCG) that acts like thyroid stimulating hormone (TSH). Thyroxin (T4) levels rise from about 6-12 weeks and peak by mid gestation, reverse changes are seen with TSH. After women give birth 20-40% are remain permanently hypothyroid.

In magnetic resonance imaging (MRI) and magnetic resonance spectroscopy (MRS) methods are used to identify changes on the brain volume or other global structural abnormalities and explains the mechanism of ID causing thyroid dysfunction. The degree of neurological impairment and the likelihood of its permanence are not only related to the severity of ID, but also to the stage of life at which the individual is exposed to it. At differing time-points, thyroid hormones have particular effects on brain maturation, regulation of neural development and microglial proliferation, dendritic arborisation, synaptogenesis, cell migration and myelination. Besides these, three other imaging techniques, nuclear imaging technique, PET and SPECT indicates that the spectrum of medical imaging techniques are to study brain changes with ID related disorders (i.e. hypothyroxinaemia, congenital hypothyroidism, hypothyroidism and cretinism) is wide.

Binocular single vision may be normal when it is bifoveal and there is no manifest deviation or it may be anomalous when the images of the fixated object are projected from the fovea of one eye and extrafoveal area of the other eye i.e.; when the foveal direction of the retinal elements has changed. A small manifest strabismus is therefore always present in anomalous binocular single vision.

Normal binocular single vision requires, clear visual axis leading to a reasonably clear vision in both eyes. The ability of the retino-cortical elements to function in association with each other to promote the fusion of

two slightly dissimilar images i.e. sensory fusion. The precise co-ordination of the two eyes for all direction of gazes, so that corresponding retino-cortical element is placed in a position to deal with two images i.e. motor fusion.

Binocular signal vision may be defining as the state of simultaneous vision, which is achieved by the co-ordinate use of both eyes. So that separate and slightly dissimilar images arising in each eye are appreciated as a single image by the process of fusion.

Thus binocular vision implies, simultaneous macular perception, in blinding of sight with some efforts and amplitude from the two eyes implies fusion and relative distance of the object from the observer called stereopsis or depth perception form, the perceptual synthesis as a single percept.

The binocular system encodes disparity, since there are several ways to represent the disparities of binocularly vision points. By measuring the difference of angles formed in two points with eyes based on triangulation and range findings system in which the smallest detectable difference in disparity found, which is the sign of depth can be correctly identified, known as sterioacuity of vision.

Motor status can be assessed by limitation of eyeballs movements or the extent of versions. So, there is measurements of angle of deviation and face turn for correcting by different measures. A further assessment needs to be made to assess the ability of the eyes to hold the convergence at the near point. This is convergence sustenance, it gives a good parameters, and called fusionalvergences.

There are varieties of sensory adaptations that occur in response to clinical situations that disrupt binocular vision. The development of a specific type of sensory adaptation depends on when (age of the patients) the sensory anomaly occurred and the severity and type of binocular disruption.

Visually immature patients may develop monofixation syndrome, anomalous retinal correspondence (ARC) or large regional suppression, where as in visually mature patients may develops diplopia, confusion or rivalry.

Amblyopia is not actually a sensory adaptation, may occurs as a consequence of suppression.

There are two mechanisms involved in the cranial nerve dysgenesis to show the strabismic disorders in which congenital non progressive ophthalmoplegia and active limitation or passive restriction of eyeglobe movement occur resulted from fibrous changes in the muscles or their tendon sheaths. One mechanism of it is the teratogenic insults to the foetal brain at the time of cranial nerve development, obviously shows that the failure of normal development of cranial nerve nuclei and their motoneurone components of cells to differentiate, aggregate and to establish proper neuronal connections. Another mechanism is involves to which genetic mutation occurs by some drugs that leads to kinesin related defects of formation of axonal transport molecules necessary for normal extraocular muscles function and development. These are the well defined clinical entities such as Duane retraction syndrome, strabismus fixus, Brown syndrome and general fibrosis syndrome.

Strabismus fixus convergence or Heavy eye syndrome (HES) is a cause of acquired esotropia in the setting of high myopia. Heavy eye syndrome is not typically seen in childhood. Patients typically have high myopia an average diopter of -18.0 D. Among their cases, some patients have complains of new onset diplopia in adulthood.

Strabismus fixus can be associated with a number of conditions including congenital fibrosis, amyloidosis and in rare occasions high myopic patients may develop a specific squint “myopic strabismus fixus” (MSF). This disease may progress over several years from a small degree of esotropia with free ocular movement to the end stage of large angle fixed esotropia.

Knowledge of eye diseases amongst the people about strabismus or squint is studied. Knowledge of eye disease is important in encouraging people to seek early treatment, which further helps in reducing the burden of visual impairment.

Strabismus can develop at any age but usually develops during childhood before 6 years of age; the peak age of onset is around 3 years. Strabismus in adulthood frequently occurs secondary to other systemic disease or mechanical damage such as trauma or brain tumour. There were studies conducted on the prevalence and psychological effects of strabismus. Globally the prevalence ranges from 2 to 6%. It has been found in study that, levels of knowledge, attitude and practice (KAP), source of information with high yearly income groups have ability to overcome this disability.

In the last 40 years, iodine deficiency and its effect on human potential, health and well being has been defined. These peoples are to be found mostly in the developing and developed countries with large populations such as China, India, Indonesia, Bangladesh, Nigeria and Zaire, but they are also found in Europe particularly in Germany and Italy.

The correction of the deficiency by use of iodized salt or iodized oil (or other means) has been shown to be followed by prevention and control of goiter and cretinism. There is also evidence of general increase in energy activity. Eyes are the visual apparatus make the power full sensory information system of human beings, which acts to gather higher form of information and knowledge inputs with the conjuncture of hearing, touch and speech modalities of sensations, requires proper effectiveness of thyroid hormones.

So that individuals and through them, the community are able to take a new interest in life and so develop a much greater degree of self reliance. Monitoring the correction of iodine deficiency is readily achieved with measurements of the dietary iodine intake through urine iodine or measurement of serum thyroidal hormones in the blood.

Compare to animal model in mammal and human being by evolution and development studies, it has been shown that, metabolic pathway activated genes controls many neurological disorders in the development and organization process of the cerebral cortex in not only associated with thyroid dysfunction but in also thyroid disrupters-goitrogens.

The three characteristic features of neurological cretinism in its fully developed form are extremely severe mental deficiency together with squint, deaf mutism and motor spasticity with disorders of the arms and legs of a characteristic nature.

To find out relationship between Hypothyroxinemia and Strabismus in the population the epidemiological surveillance and their relations were elucidate. In this thyroid disease, insufficiency of thyroid hormone thyroxin (T4) formation and its ineffectiveness is to be the basis of the development of strabismus. The interference of thyroid hormone formation occurs due to deficiency of iodine, in autoimmune thyroid diseases and varied consumption of some goitrogenic food and drugs are responsible.

Iodine deficiency varied from place to place, region to region of the country and world. According to the finding, most iodide formed in the ocean, and iodide ions in the sea water is oxidized to element iodine, after volatilization into the atmosphere which returned to the soil by rain, completing the cycle. In global worming process and climate change, this iodine cycling is slow and incomplete in many regions, so soil and ground water is deficient in iodine. Moreover leaching of iodine from soil and water occur due to deforestation, intense glaciations, river bank erosion in flooding. Animal and crops grown on these areas are deficient of iodine. Lack of consumption of iodine enhances its deficiency.

Proper consumption of iodine rich sea fish or sea vegetables in the diet or foods produced in iodine sufficient areas can overcome these deficiencies. The daily iodine requirements in children 9 to 13 years of age is 120 ug, teens 14 to 18 years and adults 150 ug , pregnant teens and women requires 220 ug.

Iodine is an essential trace element for the synthesis of thyroxin, thyroid hormone in both for mother and her developing foetus up to neonatal and postnatal period of 1000 days. Foetal neurodevelopment and potential development is only affected when the mother is hypothyroid and hypothyroxinemic in the early stages of pregnancy, even in iodine sufficient areas and the child has been suffering from iodine deficiency which needed for the thyroxin synthesis. Any situation arises which compromising thyroid hormone transfer from mother to fetus might lead to neuromuscular maldevelpment in the form of endemic goiter or cretinism with permanent lesions that contributes to postnatal development and plasticity of neural tissues.

It has been observed that identified different chemical substances and naturally occurring some food substances are recognized as goitrogens, which are usually active only if iodine supply is limited and or, goitrogen are intake in a long duration. Many of them have been tested in vitro or have been shown to possess antithyroid effects in vivo. Some goitrogens are the chemical groups acting directly on the thyroid gland. Some goitrogens are found in widely used tuber cassava. Cyanogenic glycosides or in the form of thiocyanate or isethionate, which inhibiting transport of iodide into thyroid gland. Certain sulphur containing onion volatiles, disulphide or other goitrin acting on intrathyroidal oxidation and organic binding process of iodide or coupling reaction. Excess iodide or lithium consumptions interfering with proteolysis, dehalogenation or hormone release.

Some vegetables of cruciferous family i.e. cauliflower, broccoli, cabbage, turnip or soybean have indirect goitrogenic effects to interrupts the enterohepatic circulation of the thyroid hormones. When iodine intake is limited, the interfering effects of these substances usually cannot be overcome. Thiocyanate may cross the placenta of pregnant mother and affect the thyroid of fetus.

The farmer and labourgroupof greater rural areas of the country are like to eat different goitrogen containing cruciferous vegetables or toxic weeds malvaperiflora, with onion, salt and mustard oil mixed decomposed rice in their morning meal. They become energetic with smoking, which all interferes the metabolic processes of thyroid gland and muscles.

Improper supply and deficiencies of selenium, zinc, iron, copper and vitamin A may also have goitrogenic effects. Selenium deficiency may have profound effects on thyroid hormone metabolism and possibly also on thyroid gland itself. In this situation the function of type 1 deiodinase (a selenoprotein) is impaired. Selenium deficiency also leads to reduction of selenium containing enzyme glutathione peroxidase. Glutathione peroxide detoxifies free radicle H_2O_2, which abundantly present in the thyroid gland.

An autoimmune thyroid disease (AITD) is a result of complex interaction between genetic and environmental factors. Incidence mostly in women between child bearing age 30 and 50 years. AITD genes is the HLA-DR gene locus as well non–MHC genes including thyroglobulin (Tg) and thyroid stimulating hormone (THR) receptor genes.

The major environment triggers are excess iodine, medication of some drugs i.e. iodine containing drugs, amiodarone, infections like rubella and hepatitis c virus acts as indirect precipitators, smoking , stress, sex steroids and with pregnancy.

Diagnosis is based on clinical features and supported laboratory investigations. There is a weak linear correlation between thyroid receptor antibodies with the titre of antibodies against different serotypes of Yersinia enterocolitica antigens. It can be considered that Y. enterocolitica infections may play a role in etiology of Graves' disease. Thyroid and intestinal diseases prevalently coexist- Hashimoto's thyroiditis (HT) Grave's disease (GD) are the most common autoimmune thyroid diseases (AITD) and often co-occur with celiac disease (CD) andnon celiac wheat sensitivity (NCWS). This can be explained by the damaged intestinal barriers and the following increase of intestinal permeability, allowing antigens to pass more easily and activate the immune system or cross reacts with extra intestinal tissues repeatedly (Leaky Gut Syndrome).

Additionally, the composition of the microbiota has influence on the availability of essential micronutrients for the thyroid gland. Iodine, Iron, Copper are crucial for thyroid hormone synthesis, Selenium and Zinc are needed for converting T4 to T3 and Vitamin D assists in regulating immune response. These micronutrients are often found to be deficient in AITDs resulting in malfunctioning of the thyroid.

The gut microbiota largely regulates homeostasis as well as development of gut associated lymphatic tissue (GALT). The immune system itself has an influence on the composition of the intestinal microbiota, which underlines the symbiotic relationship. Deiodinase activity has been found in the intestinal wall and could contribute to total T3 body levels. Another influencing factor of microbiota is its effects on neuro transmitters such as, Dopamine, which can inhibit thyroid stimulating hormone (TSH).

Knowledge of eye disease amongst the people about strabismus or squint is studied. Knowledge of eye disease is important in encouraging people to seek early treatment which further helps in reducing visual impairment like strabismic amblyopia, a burden of family and society. Studies in Ethopia showed that, the level of good knowledge in the population was 37%, compare to the other region of the world. In India 94.7% of participants had known the consequences of strabismus, in Jeddah (Saudi-Arabia) 75% of participants know etiologies, in Nigeria 50% participants did not know, what is amblyopia or squints.

It has been found in comparative studies that, variable picture of depth of knowledge, source of information and socio-economic conditions imposed to pile the problem.

The problems hypothyroxinemia leading strabismus and developmental consequences of eye problems is an inconsistent impediment from view of maximum effectively as binocular vision can be ensured by the different treatment and therapeutic measures. The successful management of these cases adopted according to the stages of complications, age of the patients, period of time when they notices , their accessibility in spot and capability to take into action. However, the searching cases of strabismus and its forms associated hypothyroxinemia carefully analyzed, and the considering materials would be the basis to form the useful empirical principles, from which necessary therapeutic guidelines can be introduced.

So, this is a desirable thing from the view of maximum effectivity can be ensured especially in younger age group.

By taking estimating and reliable correcting measures with lens, prisms, orthoptics, drugs, anti-suppression, chemical denervation or surgery of the extra ocular muscles.

RECOMMENDATIONS:

1. Take the appropriate measure to ensure the quality and appropriate iodine valued iodized salt nationwide to its retailers and to its every household consumer respectively.
2. Attains the good knowledge, attitude and practices not to consume decomposed, over dated-contaminated, goitrogenic vegetables and food goods brought from market.
3. Ensure the surveillance and assessment programs from time to time for eye examination and vision testing of school children and antenatal and postnatal checkup of mother and her child, including thyroid functions.
4. Take easy cost effective controlling measures taken earlier than by giving high compensating management values in late vision depriving intellectual eye complications.
5. Develop a common consensus, frame the direction, and build our awareness so that, the mitochondrial powerhouse will not be energetic or carry on its functions properly unless we supply a few value added iodine in our daily diet on the priority basis of the consideration.

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