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Review Article

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Autoimmune and Non-Autoimmune Thyroid Diseases and Type 1 Diabetes

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Introduction

Type 1 diabetes results from the destruction of Langerhans β-cells by autoimmune processes. Thyroid disease and type 1 diabetes are often the two most common endocrine disorders encountered in clinical practice. In around 30% of cases, T1DM is associated with other organ-specific autoimmune diseases. These are most often thyroid disorders (Graves' disease and, above all, Hashimoto's thyroiditis), which are three times more frequent in type 1 diabetics than in the general population [1]. The high frequency of thyroid pathologies in patients with T1DM prompts the need for systematic screening for these conditions, using highly specific circulating thyroid antibodies and hormones (anti-TPO, anti-TG, FT3, FT4, TSH).

In the ISPAD 2000 consensus guidelines, the recommendations for thyroid disease are as follows at the time of diagnosis of diabetes, thyroid function tests and anti- thyroid antibodies should be performed to detect asymptomatic thyroid disease. Many centers use thyroid function tests as part of an annual check-up [2,3].

The aim of our literature review is to determine the prevalence of thyroid pathologies in subjects with type 1 diabetes.

Pathophysiology of type 1 diabetes

Immunology of Diabetes

In a susceptible genetic background, a triggering factor, probably environmental, is responsible for a breakdown in immune tolerance. A series of arguments point to the involvement of the immune system in the pathophysiology of type 1 diabetes. Insulitis, the infiltration of the islets of Langerhans by mononuclear cells within the pancreas, is the anatomical hallmark of the disease. The immune response thus initiated leads to the production of autoantibodies and the activation of cytotoxicity mechanisms responsible for β- cell destruction.

Insults

Insulitis, an inflammatory infiltrate of the islets of Langerhans, is the histological hallmark of type 1 diabetes. Historically, it is also the first argument in support of an autoimmune mechanism in the pathophysiology of the disease. Data on insulitis in humans are mainly derived from early autopsy studies carried out just after the discovery of diabetes. Insulitis is composed of LT with a dominance of CD8+ LT; CD4+ LT; macrophages and rare B lymphocytes.

Cellular mechanism [4]

The predominance of T-lymphocytes in insulitis, the demonstration of T-lymphocyte autoreactivity to insular antigens in T1DM, and the partial efficacy of T-lymphocyte-targeted immunointervention in humans all point to a central role for these cells in the pathophysiology of the disease. Several studies have highlighted the role of these autoreactive T lymphocytes in the death of pancreatic beta cells. Recent work by the« immune mechanisms of type 1 diabetes» team (Inserm/University Paris Descartes) in NOD mice, a model used to study type 1 diabetes, reveals the essential role of innate immune system cells which had not previously been implicated in diabetes. In this study, the researchers succeeded in describing the mechanisms that initiate the activation of T lymphocytes directed against pancreatic beta cells (figure 1).

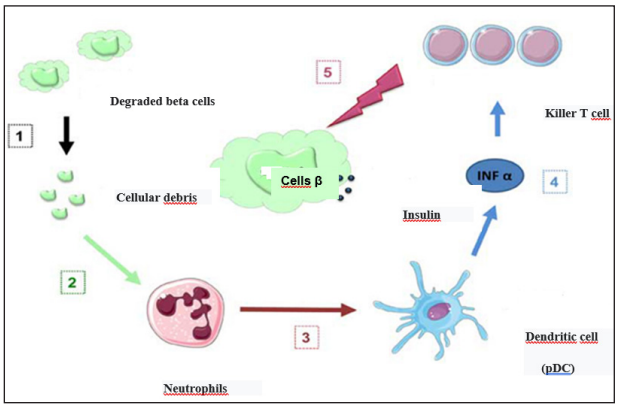

Figure 1: immune system activation leading to β-cell death

The natural degradation of β-cells (1) leaves cellular debris in the tissues, which abnormally activates neutrophils (2).

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These immune system warning cells inform dendritic cells (pDCs) (3), which in turn trigger the production of interferon IFN α, a warning molecule (4). Interferon α then stimulates T lymphocytes which, by recognizing functional pancreatic β cells, induce the death of these cells (5). Insular autoantigen-specific CD8+ T cells have also been detected in the periphery of human diabetes. The cytolytic action of CD8+ LTs is exerted by various pathways inducing apoptosis of the target cell: perforin and granzyme production, interaction between the Fas molecule expressed by the LT and its ligand (FasL) expressed by the target cell, secretion of TNFα.

Nature of the Auto-Antigen [1]

Several autoantigens have been identified, based on their recognition by antibodies or autoreactive T cells during the preclinical and early clinical phases of T1DM. However, the autoantigen responsible for triggering the immune response against pancreatic beta cells has not yet been formally identified in humans, but proinsulin remains the best candidate.

Until now, insulin and proinsulin have been considered the only β-cell-specific autoantigens. In humans, anti-insulin antibodies are the first to appear during the preclinical phase in children, and are strongly associated with progression to diabetes. Anti-glutamate decarboxylase (GAD) antibodies, an enzyme expressed in the brain and β-cell, are present in almost 80% of insulin-dependent diabetic children at the onset of the disease, and in 3% of first-degree relatives. Numerous other autoantigens, recognized by antibodies and/or T lymphocytes, have been described in T1DM. These include thyrosine phosphatase IA-2, anti- GLIMA38, anti-Znt8 and anticarboxypeptidase H, but are less frequently present.

Genetic Factors [1,3]

Type 1 diabetes is a heterogeneous disease with polygenic inheritance. The hereditary nature of T1DM most often translates into an increased risk of the disease in those with first- degree relatives (parent, sibling, child) of a type 1 diabetic subject, compared with the general population. The prevalence of the disease is around 10% to 13% in children whose families include an affected subject, while the prevalence of diabetes in the general population is 0.3%. The intensity of this individual risk varies according to the type and number of affected relatives (Table 1). The higher concordance rate for T1DM in monozygotic twins (on average 33%) than in dizygotic twins (10-20%), plus the presence of familial aggregation of the disease in 10% of cases, testify to the intervention of genetic factors of susceptibility to type 1 diabetes. Its transmission does not follow a Mendelian model, but is complex, reflecting its multigenic character, and it is accepted that the disease results from the effect of different genes interacting with each other and contributing to genetic susceptibility in different individuals.

Table 1: Absolute risk of diabetes for a first-degree relative of a diabetic subject.

Diabetic patients	Risk
Father	6% (for her child)
Mother	2% (for her child)
Father and mother	30% (for their child)
Brother or sister	5% for siblings)
Monozygotic twin	33% (for his twin)
Two people affected	30%
General population	0.3%

The study of genome-wide polymorphic markers in diabetic subjects has identified around twenty genetic regions associated with susceptibility to the disease, the main ones being the HLA class 2 complex gene and the insulin gene. Other genes have been implicated in the disease, but with a lower risk than the HLA complex genes (Figure 2).

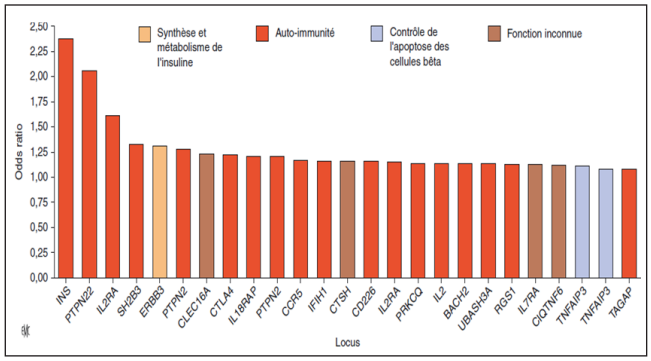

Figure 2: function of genes thought to be associated with T1D risk

Environmental Factors

The 30-70% concordance rate for disease in monozygotic twins, and the development of type 1 diabetes in only 10% of individuals with a genetic predisposition, point to the existence of non-genetic factors in disease determinism [1-5]. Thus, the marked geographical variations in the annual incidence of T1DM and the increased prevalence of the disease in populations migrating from a low-incidence region to a high-risk region support the crucial influence of the geographical environment.

Environmental factors may intervene at different stages of the disease, with variable effects depending on the stage of intervention.

Infectious Agents [1,5-7]

The link between viral infection and T1D is based on epidemiological and therefore indirect arguments, and essentially concerns enteroviruses, more specifically Coxsakie B virus. However, infectious agents may have protective effects on the development of T1DM, explained by the fact that the reduced stimulation of innate immunity, due to a lower prevalence of pathogens, would induce a maturation bias in the immune system, favoring the development of atopy and autoimmunity.

Dietetic Factor [6]

Dietary factors can, in certain circumstances, influence the development of type 1 diabetes. It has been shown that children fed cow's milk during infancy are more likely to have developed type 1 diabetes than those not breastfed by their mothers, or only briefly, particularly in subjects carrying the HLA haplotype for susceptibility to T1DM. The early introduction of cereals into the diet, irrespective of their gluten content, has also been associated with an elevated risk of β-cell autoimmunity.

Minor hypoglycemia is difficult to avoid when the metabolic balance corresponds to a low risk of severe microangiopathy. In the case of major hypoglycemia, the frequency of occurrence is higher the lower the HBA1c, particularly for values below 6% (figure3).

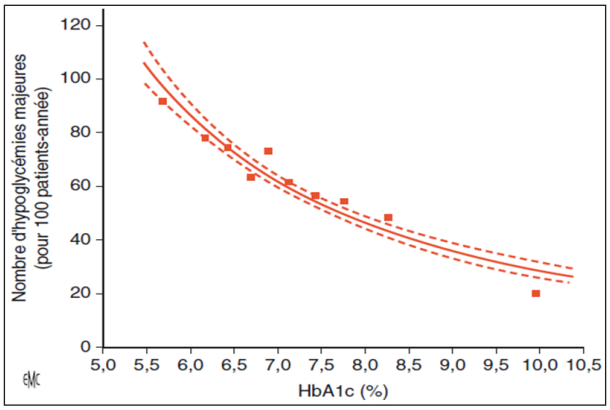

Figure 3: Frequency of severe hypoglycemia as a function of glycated hemoglobin (HbA1c)

Etiology

The search for a causal factor is classic (Table 2). These factors are rarely the cause of repeated major hypoglycemia. Common factors are: long-standing diabetes (more than 9 years), a history of severe hypoglycemia, a rapid fall in HbA1c or a rapid increase in insulin doses

Table 2: Classic Causes of Hypoglycemia

Excessive insulin dose
Insulin absorption too rapid
Eating disorders
Endocrine disease
Digestive disease
Liver disease
Anti-insulin antibodies
Long physical effort
Products that modify the perception of hypoglycemia

Treatment Monitoring

Capillary Blood Glucose Measurement

From one consultation to the next, blood glucose levels are used to determine the patient's blood glucose profile. Despite variations in values from one day to the next, it is often possible to discern trends.

Glycated Hemoglobin HBA1c

HBA1c is the essential criterion for monitoring glycemic control in diabetic patients. It reflects the average blood glucose level over the 3 months prior to dosing (Figure 4). Values in excess of 8% are indicative of insufficient insulin dosage and/or diet errors, while HBA1c values in excess of 10% are indicative of missed injections and diet failure

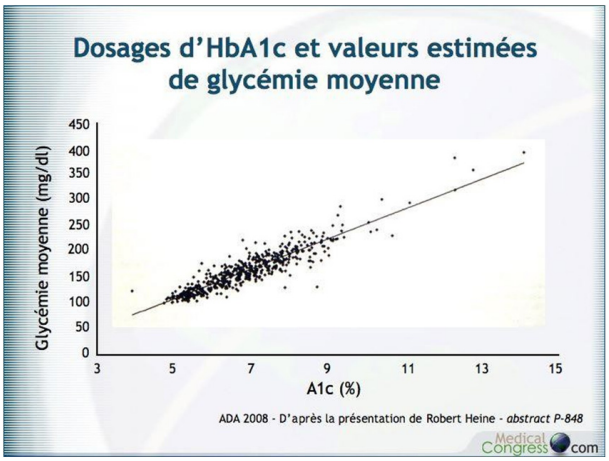

Figure 4: HbA1c assay and estimated mean blood glucose values

Thyroid Pathologies in Children

Thyroid Physiology [11,12]

Thyroid Hormone Biosynthesis

Hormone biosynthesis takes place in the thyroid in successive stages, regulated by TSH and iodine intake.

The basic ingredients of thyroid hormone synthesis are tyrosine and iodine, both of which are taken from the blood by follicular cells.

All stages of thyroid hormone synthesis take place in the thyroglobulin molecule of the colloid. Tyrosine is incorporated into the large thyroglobulin molecule during its synthesis. Once formed, the tyrosine-containing thyroglobulin molecule is exported by exocytosis to the follicular cavity colloid.

The iodine required for synthesis comes from food, and is converted to **I-** in the stomach. Follicular cells then capture **I-** ions in an active process driven by a concentration gradient. This step involves the Na+ K+ ATP**ase** and the Na/I or NIS symporter. In the colloid, the active iodine is then organified on the tyrosyl residues of thyroglobulin (**Tg**), generating the formation of **mon-iodotyrosine (MIT)** and **diodotyrosine (DIT)**.

This is followed by the coupling of iodinated tyrosines to form thyroid hormones. The coupling of a DIT and a MIT gives rise to **triiodothyronine** or **T3**, while the coupling of two

DITs gives rise to **tetraiodothyronine (T4 or thyroxine)**. The products of these chemical reactions remain bound to thyroglobulin, where they are stored until released for secretion.

The Role of Thyroid Hormones

Virtually every tissue in the body is influenced directly or indirectly by thyroid hormone.

- o Effect on Metabolic Activity and Heat Production:
- Increases basic metabolism.
- Main factor determining O2 consumption and resting energy expenditure.
- Calorigenic effect: increased metabolism goes hand in hand with increased heat production.
- o Sympathomimetic Effect:
- Increases target cell response to catecholamines, noradrenalin and adrenalin.

o Effect on the Heart:
- Enhances the effect of catecholamines on the heart.
- Speeds up the heart and stimulates its contraction force, thus increasing cardiac output.

o Effect on Growth and the Nervous System:
- Stimulates somatotropic hormone (TSH) secretion and promotes its action on skeletal growth and protein synthesis.
- Crucial role in the normal development of the nervous system, especially the CNS.
- Important roles in the normal functioning of the adult CNS.

Regulation of Thyroid Hormone Secretion

Thyroid hormone secretion depends on the hypothalamic-pituitary axis. Thyrotropic hormone (**TSH**), a thyroid-stimulating hormone secreted by the anterior pituitary gland, is the main factor on which thyroid hormone secretion depends. In addition to stimulating thyroid hormone production, **TSH** is responsible for keeping the thyroid gland in good condition. In the absence of **TSH**, the thyroid gland atrophies and secretes very little hormone. Conversely, it hypertrophies and hyperplasifies when over-stimulated by **TSH**. Hypothalamic **thyrotropin** releasing hormone (**TRH**) stimulates **TSH** production by the anterior pituitary, while thyroid hormone exerts a negative feedback which reduces it. As with other negative feedbacks, this tends to stabilize thyroid hormone production (Figure 6).

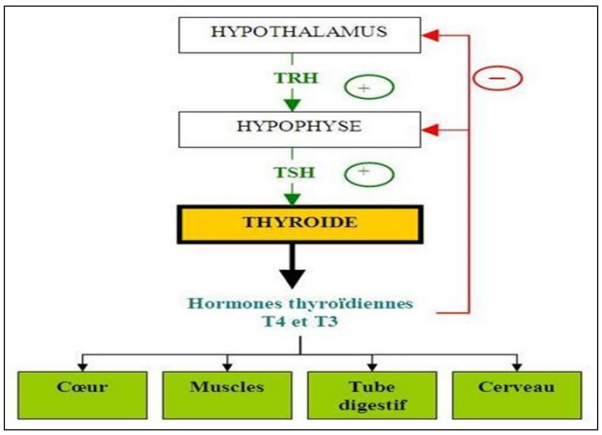

Figure 6: regulation of thyroid secretion

Exploration of the Thyroid Gland [13-15]

Thyroid Hormone Assay

Thyroid hormone assays have improved considerably over the last twenty years. Today's assays boast excellent sensitivity and specificity, thanks to the use of monoclonal antibodies and improved detection signals. The methods used were competitive radioactive assays, increasingly replaced by automated immunometric assays using enzymatic or luminescent tracers.

Thyrotropin TSH:

TSH is the most valuable parameter for assessing thyroid function. It is the parameter of first choice.

TSH concentrations vary throughout the day. Accepted values in Europe range from 0.4 to 4mUI/L for ambulatory subjects. Recent studies have shown the variability of the upper limits of this assay, depending on a number of parameters such as BMI, presence of antithyroperoxidase antibodies, diabetes, hypertension, etc., and the assay methods used.

For hospitalized patients, normal TSH values are wider, ranging from 0.05 to 10 mIU/l.

T3 and T4 total, T3 and T4 free:

Thyroxine (T4) makes up around 90% of the hormones secreted by the thyroid, yet its biological activity is about four times less than that of T3. Its concentration is an excellent reflection of thyroid production. T4 circulates in the blood in free form (0.02%) and bound to carrier proteins (albumin, transthyretin and TBG). Around 80% of circulating T3 comes from T4 deiodination in peripheral tissues (liver, muscle, brain, etc.). T3 and T3L reflect peripheral production, and their diagnostic value in assessing thyroid function is limited. Assays of free forms have supplanted those of total hormones, which are too dependent on changes in transport protein concentration or affinity (pregnancy, estrogen treatment, renal failure). These assays are now automated.

Other Dosages:

Anti-TSH Receptor Antibodies:
- Pathognomonics of Graves' disease
- Can be stimulating or blocking
- To be measured in cases of clinical suspicion only

Anti-peroxidase antibodies (anti-TPO):
- Can be useful in confirming the diagnosis of autoimmune thyroiditis
- High positivity in elderly populations

Anti-thyroglobulin antibodies:
- Less specific than anti-TPO because frequent positivity in the general population.

Scintigraphy

This examination is a functional image of the thyroid gland. It shows whether certain areas of the gland are more functional than others. It is performed mainly in cases of hyperthyroidism, particularly if there is a goiter or nodules. In practice, this examination is carried out by injecting a very small quantity of radioactive material (Technetium 99m) into the vein, which is rapidly eliminated from the body (approximately 48 hours).

After a delay of around 30 minutes, the thyroid is imaged using a gamma camera.

Ultrasound

The aim is to measure very precisely the dimensions of the thyroid gland and define its structure. It determines whether or not there are nodules, and whether they are solid or liquid (cysts). Its performance enables the detection of very small lesions, as small as 2 to 3 millimeters. In practice, the examination is painless and of short duration. After applying a gel to the base of the neck, an ultrasound probe is passed over the area to be explored. The echoes are recorded on a screen and then photographed (figure 7).

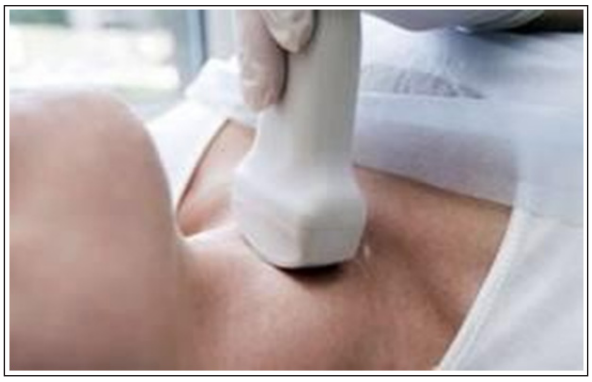

Figure 7: Thyroid ultrasound

Cytopuncture
It is performed to complete the exploration of certain nodules or to empty cysts. Microscopic analysis of the cells removed enables us to distinguish common nodules from those requiring surgical treatment. This examination is performed by removing a small quantity of cell- containing liquid through the skin, using a very fine needle. To optimize the quality of the examination, three to four different punctures are made into the nodule.

Thyroid function abnormalities in children
Abnormalities of thyroid gland function are among the most common endocrine disorders. They fall into two broad categories, **hypothyroidism** and **hyperthyroidism**, depending on whether there is a deficiency or excess of thyroid hormone secretion respectively. The consequences of thyroid hormone deficiency or excess are largely predictable from the known physiological effects of this hormone.

Hyperthyroidism [16-20]
Introduction
Hyperthyroidism is defined as hyperfunction of the thyroid gland, leading to increased hormone production and a state of thyroid hormone intoxication (or thyrotoxicosis). It is often less common than in adults, but increases in frequency during adolescence, when the picture is little different from that of adult Graves' disease. The prevalence of clinical hyperthyroidism is traditionally estimated at between 0.5% and 2% of the adult population, with a 10-fold predominance in women. Fetal and neonatal hyperthyroidism is rare, with a prevalence of 1 in 50,000 newborns.

Etiopathogenesis
Pathogenesis in children is the same as in adults

Graves' Disease
Graves' disease is the most common cause of hyperthyroidism. It occurs voluntarily in a family context of thyropathy, with a predilection for young women, but does not spare children, men or the elderly. In newborns, it may be observed transiently, due to the transplacental passage of thyroid-stimulating immunoglobulins (Ig).

- **Pathophysiology:** It results from the production of thyroid-stimulating Ig by intra- thyroid lymphocytes. It occurs in a genetic predisposition where class 2 tissue antigens are expressed on the surface of thyrocytes. Anti-TSH receptor autoantibodies are IgG antibodies that cross the placental barrier. In genetically predisposed individuals, Graves' disease can be triggered by environmental factors such as stress, steroid hormones, smoking, high dietary iodine intake or immunomodulators such as interferon alpha.
- **Clinical presentation and diagnostic evaluation:** Graves' disease is often uncommon in childhood. Symptoms include increasing difficulty concentrating, increased intestinal transit, nocturia, fatigue and fatigability on exercise, in an often thin child. A goiter, usually symmetrical and smooth, with a firm or soft consistency. Thus, hyperthyroidism should be sought in children studied for tachycardia, weight loss, dyspnea, or attention deficit/hyperactivity disorder. Ocular signs are less pronounced than in adults.

TSH is suppressed, and T4 and T3 levels are usually elevated, but sometimes only T3 is elevated. Thyroid scintigraphy is usually unnecessary for diagnosis.

Ultrasound can be useful to rule out a toxic nodule, particularly if the goiter is small

Treatment of Graves' Disease
The treatment of Graves' disease is not straightforward, since it requires a choice between three modalities: synthetic antithyroid drugs (ATS), surgery or radioiodine. Each of these three treatments has its own particular indications, side effects and failures. The choice of treatment depends on the patient's age and the size of the goiter, and compliance is particularly difficult in adolescence.

Hashimoto's Thyroiditis
Very rarely, the first phase of thyroiditis may give rise to thyrotoxicosis (Hashitoxicosis). The presentation is peculiar: very firm, non-vascular goiter; ophthalmopathy, even pretibial myxedema; presence of high titers of anti-thyroperoxidase and anti-RTSH antibodies; increased iodine-123 uptake; early progression to hypothyroidism, sometimes with gland atrophy.

Secondarily Toxic Multinodular Goitres
It encompasses a spectrum of different clinical entities, ranging from a single hyperfunctioning nodule to multiple hypo- or hyperfunctioning nodular areas within a goiter. The mechanism of hyperfunction has recently been characterized as somatic activating mutations of the TSH receptor, which are implicated in the pathogenesis of GMN (multinodular goiter) as in toxic nodules.

GMN is the main cause of hyperthyroidism in the elderly, occurring on a pre-existing goiter whose nodules become autonomous. The goiter is diffuse, often irregular and bumpy, distorted by the presence of nodular formations, and sometimes associated with compressive signs.

Toxic Nodule
The toxic nodule is a benign monoclonal tumor whose growth and function escape pituitary control. In iodine-deficient regions, nodules are often due to activating mutations in the TSH receptor or Gs α protein. The presence of TSH receptor mutations is not constant (varying from 10% to 80% depending on the technique used and the degree of iodine deficiency).

Activating mutations in the Gs α protein occur in around 10% of cases.

The nodule represents a localized hypertrophy of part of a lobe, but may not be clinically perceptible. Thyroid assays confirm hyperthyroidism and the absence of antithyroid autoimmunity. Thyroid scintigraphy is the fundamental diagnostic tool.

Iatrogenic Hyperthyroidism

Iodine Overload: iodine-containing drugs (cough suppressants, antidiarrheals, amiodarone), iodine-containing contrast agents, iodine-containing antiseptics or iodine-rich food preparations. They may be transient, or sometimes prolonged and severe, especially with amiodarone.

Lithium Interferon: lithium affects cellular function via its inhibitory action on adenosine triphosphate (ATPase) and cAMP. It is concentrated in the thyroid gland and can lead to intra-thyroidal iodine overload. Lithium also inhibits iodotyrosine coupling, alters the structure of Tg, inhibits Tg proteolysis and thus hormone secretion. The onset of hyperthyroidism may be explained by intra-thyroidal retention of iodine.

Treatment

Identifying the cause of hyperthyroidism is an essential prerequisite for choosing the right treatment. Hyperthyroidism is a chronic condition, and initiation of therapy is not an emergency.

In most cases, treatment consists of inhibiting thyroid production (antithyroid drugs), or reducing functional parenchymal mass (surgery, metabolic radiotherapy).

We will present the different treatments for each of the hyperthyroidisms.

Toxic Nodule: surgery is a safe and traditional treatment for toxic nodules, usually considered after reduction of hyperthyroidism with medication.

Hyperthyroidism disappears with 131 iodine in 85 to 100% of cases.

Secondarily Toxic Multinodular Goiter: medical treatment is not curative, and is only useful in preparation for radical treatment: total or subtotal thyroidectomy, or radioiodine in the elderly.

Hashimoto's Thyroiditis: beta-blockers can be used to treat thyrotoxicosis symptomatically. Radical treatments should be avoided, as hypothyroidism develops rapidly within a few months, and atrophy of the gland is common.

Hyperthyroidism of Iatrogenic Origin: In the case of lithium, thyrotoxic states linked to intra-thyroidal iodine accumulation and cytolysis may simply benefit from betablockers. If there is hyperfunctional activity due to Graves' disease, antithyroid treatment is justified.

In the event of iodine overload, particularly induced by amiodarone, it is advisable to discuss with the cardiologist whether the product in question should be discontinued. Functional hyperthyroidism in nodular parenchyma (type 1) is treated with ATS, whereas a lesional mechanism requires the prescription of corticoids (type 2).

Conclusion

Hyperthyroidism causes general discomfort and can lead to complications, particularly of a cardiac or skeletal nature. The cause of hyperthyroidism has a major influence on prognosis and choice of treatment.

Hypothyroidism in children [17,18]

Introduction

Hypothyroidism refers to hypofunction of the thyroid gland, resulting in reduced hormone production and a state of hypometabolism. It is of particular concern in children, as thyroxine plays a decisive role in brain growth and differentiation, which begins in utero and continues after birth.

Hypothyroidism is one of the most common endocrine disorders, with an estimated prevalence of between 1% and 2% of the population. It is more common in women than in men (sex ratio 1:10), and its incidence increases with age, mainly after the menopause. The frequency of hypothyroidism in newborns is estimated at around 1 per 3,5000 to 4,000 births.

Etiopathogenesis

T4 deficiency acts differently depending on the child's age. These effects reduce cell multiplication and nerve connections at birth, while at the same time inhibiting growth in height and weight. Hormone deficiency also leads to hypometabolism, slowing down all vital functions.

Congenital hypothyroidism in newborns is primarily due to thyroid dysgenesis: 85% have thyroid dysgenesis, of which 80% are ectopic, and 20% have agenesis of the gland (athyreosis). The remaining 15% of cases are due to a hormone-synthesis disorder, often undetected at birth.

Acquired forms are primarily autoimmune, sometimes metabolic or lesional. Secondary forms (hypothalamohypophyseal) are part of multiple hypopituitarism, with a few isolated cases linked to mutations in TSH beta subunits.

Malformative hypothyroidism recognizes a multifactorial origin: genetic: proven by the predominance of females and some ethnic variations, but the presence of familial cases of morphogenesis disorders limited to 2% is an argument for incomplete Mendelian transmission.

Auto-immune: in 50% of cases, cytotoxic or cell-growth-blocking antibodies have been identified.

Environmental: toxic factors or viral infections, but there are no consistent arguments in favor of an important role for environmental factors.

Clinics

The clinical signs of hypothyroidism are :

Congenital

- Particular facies: nasal ensellure, macroglossia, abundant hair,
- Dry, mottled skin, persistent neonatal jaundice,

- Fontanelle very wide, posterior fontanelle open,
- Abdominal distension, umbilical hernia,
- Hypotonia, hypoactivity,
- Constipation,
- Difficulty sucking, hoarse crying.

Acquired

- Fatigue
- Weight gain and slower statural growth,
- Frizziness, dry skin,
- Constipation,
- Delayed bone age

Complication

Staturales: forms diagnosed in the prepubertal period may be complicated by short stature due to accelerated pubertal development, when treatment is initiated, raising the issue of a treatment to slow puberty.

Psychological: forms recognized late were accompanied by more or less severe debility, in line with the severity of the disease and the delay in diagnosis. With screening, the debility has disappeared, while forms treated early (around 1 month) may have some minor repercussions: learning difficulties in mathematics, written or spoken language disorders, fine motor skills or spatialization disorders.

Processing

The treatment of choice is daily administration of thyroxine. The full dose can be used from the outset in children and adolescents. In µg per kilogram, it is around 5 for the 1-5 age group, 4 for the 6-12 age group and 3 for adolescents.

In addition to clinical monitoring (growth and bone maturation must be normal), subsequent treatment monitoring is essentially biological: TSH and free T4, subsequently assessed twice a year, must remain within the high normal range for T4 and always normal for TSH, by adjusting the dosage of L-thyroxine ®.

Conclusion

Thyroid dysfunction in children is highly diverse, with varying effects on brain development, growth and metabolism throughout life. It is vital to know the precise etiology of dysthyroidism in order to define the most appropriate therapeutic management.

References

1. Dubois-Laforgue D. Etiologie et physiopathologie du diabète de type 1. EMC (Elsevier Masson SAS, Paris), Endocrinologie-nutrition. 2007.
2. Officinal. diabetes « l'épidémie » silencieuse ,l'officinal N90, janvier/février. 2012.
3. Tony Merriman. Vers une compréhension des causes génétique du diabète. Revue Diabetes Voice. 2004. 49: 1.
4. Bouhours-Nouet N, Coutant R. Aspects cliniques et diagnostiques du diabète de l'enfant. EMC (Elsevier Masson SAS, Paris), Pédiatrie. 2011.
5. Brilot F, Geenen V. le rôle des virus dans la pathogénie du diabète de type 1. Rev Med Liège 2005. 5: 297-302.
6. Danièle Dubois-Laforgue, José Timsit. Diabète de type 1 et environnement. 2000. 16.
7. José Timsit, Christian Boitard, Etienne Larger. Etiologie et physiopathologie du diabète de type 1. EMC - Endocrinologie-Nutrition 1996: 1- 10.
8. Julien Diana, Yannick Simon. Le rôle des cellules immunitaires innées dans le développement du diabète de type 1.Inserm Paris. 2012.
9. Kury Paulin S, Cachot V, Perfornis A, Cétoacidose diabétique. EMC (Elsevier masson SAS Pari; Endocrinologie-Nutrition. 2007.
10. Boileau P, Merle B, Bougneres PF. Traitement du diabète de l'enfant et de l'adolescent. EMC-Pédiatrie. 2005. 2 : 163-178.
11. Berthet J, Amar-Costesec A. physiology humaine ,2nd édition e Boeck Larcier. 2006. 547-552.
12. Massart C Ecorbineau. Transpoteurs d'iodures et fonction thyroïdienne. Elsevier SAS. 2006.
13. Ellakhdi FE, Naamane A. Thyroid hormone balance: recommandations and technical plug for interpretation of changes and traps. Les technologies de laboratoire. 2010. 5 : 21.
14. Centre Paul strauss. Mars. 2012.
15. Michelle d'Herbomeza. Exploration biologique de la thyroide.Revue Francophone des laboratoires. 2009.
16. Proust-Lemoine E, Wémeau J-L. Hyperthyroidie. EMC (Elesevier Masson SAS,Paris): Traité de Médecine Akos. 2009. 12 : 1.
17. Toublanc JE. Pathologie thyroïdienne de l'enfant (hypothyroïdie, hyperthyroïdie et cancer). EMC (Elsevier Masson SAS,Paris): Pédiatrie. 2008.