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Review

Iodine deficiency: Physiological, clinical and epidemiological features, and pre-analytical considerations

Carence en iode : caractéristiques physiologiques, cliniques et épidémiologiques et considérations pré-analytiques

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Abstract

Low dietary intake is associated with severe pathologies (especially goiter and cretinism) that affect life quality. Iodine deficiency disorders are a major public health problem worldwide. In fact, 246 million school-aged children have insufficient iodine intake (data from 2012). Extrapoling this value to general population leads to the estimation that 1.9 billion people have insufficient iodine intake. So, it is crucial to interpret correctly data from iodine status survey. The World Health Organization recommends urinary iodine as the main indicator for the assessment of iodine status in epidemiological surveys. To improve the result, some considerations can be taken into account by the biologist, epidemiologist or public health physician for the realization of epidemiological surveys. After a reminder about the physiological and physiopathological feature of iodine, a description of some useful parameters was made to improve the exploration of iodine status in epidemiological surveys.

Keywords: Urinary iodine; Thyroid hormones; Iodine deficiency disorders; Endemic goiter; Epidemiological survey

Résumé

La carence en iode est associée à des pathologies sévères, telles que le goitre ou le crétinisme, affectant la qualité de vie. Ce déficit nutritionnel constitue un problème mondial de santé publique. En effet, environ 246 millions des enfants de 6 à12 ans sont carencés en iode, ce qui représente 1,92 milliards de personnes de la population mondiale. Il est donc important d'interpréter correctement les données sur le statut en iode. L'organisation mondiale de la santé recommande le dosage de l'iode urinaire comme principal paramètre pour l'exploration du statut nutritionnel en iode au cours des enquêtes épidémiologiques. Cependant, des précautions sont à prendre en considération par le biologiste, l'épidémiologiste ou le médecin de la santé publique lors des enquêtes épidémiologiques pour l'optimisation de la précision des données. Après un rappel des aspects physiologiques et physiopathologiques de l'iode alimentaire, une description des paramètres utiles à l'amélioration de l'exploration du statut en iode dans enquêtes épidémiologiques a été présentée.

Mots clés : Iode urinaire ; Hormones thyroïdiennes ; Troubles dus à la carence en iode ; Goitre endémique ; Enquête épidémiologique

1. Introduction

Iodine is an essential trace element for thyroid hormones synthesis. Inadequate iodine diets may promote a deficiency that affects the thyroid's function. It is well known that iodine deficiency (ID) may occur especially in remote areas, promoting the appearance of impairments that affect all age groups from fetus until adult age. ID is one of the principal causes of impaired cognitive development in children. Cretinism is the most severe form of cognitive impairment, which can be observed due to ID during pregnancy. These nervous diseases are well characterized today and are irreversible. Some other undesirable facts

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Table 1
Recommended dosages of daily iodine supplementation (adapted from[2]) (with WHO permission).

	Daily dose of iodine supplement (μg/day)
Children < 2 years[a,b]	90
Women of reproductive age (15–49 years)	150
Pregnant women	250
Lactating women	250

[a] For children 0–6 months of age, iodine supplementation should be given through breast milk. This implies that the child is exclusively breastfed and that the lactating mother received iodine supplementation.

[b] These figures for iodine supplements are given in situations where complementary food fortified with iodine is not available, in which case iodine supplementation is required for children of 7–24 months of age.

may occur like prenatal mortality, low birth weight, and low intellectual quotient. Since 1990, the World Health Organization (WHO) has coordinated an international program for the prevention from ID in a special resolution WHA43.2. In 1993, WHO, UNICEF and International Council for the Control of iodine deficiency (ICCIDD) have adopted iodized salt as the main strategy for the control of ID. About 1.92 billion people suffer from ID throughout the world that is why it is considered as a worldwide health problem. This exploration of iodine state was possible by measuring the urinary iodine (UI) concentration in school-aged children, which allows to an extrapolation in the general population. However, there are some remaining limits to the utilization of UI data from school-aged children, such as the non-representativeness of outcome in pregnant women. Some pre-analytical considerations may affect the predictability of the result like time of sampling, delay since last meal and sampling, and the means of result expression.

2. Iodine physiology

Dietary process is the major source of iodine intake: seafood (40–320 μg/100 g), fish (25–75 μg/100 g) [1]. The most vulnerable groups, children 7–24 months of age, pregnant and lactating women, should be supplemented with iodine if iodized salt is not accessible. In cases where it is difficult to reach pregnant women, supplementation to all women of reproductive age is advised (Table 1) [2].

Iodine is essential for thyroid hormone biosynthesis. In general, this anion trace element exists as iodine in the diet and it is converted to iodide anion (I^-) in gastric mucosa. Its absorption is operated rapidly throughout the small intestine especially across the enterocytes. Iodine metabolism is closely related to thyroid hormones synthesis. A certain amount of inorganic iodide is actively cleared from blood plasma by thyroid gland and stored in the lumen of follicular cells as iodine compound. About 90% of ingested iodide is excreted in urine [3]. Iodide transport by thyrocytes is a two-step process involving basal and apical transmembrane transporters. The active co-transporter exists into the basolateral membrane of thyroid gland cells known as NIS (sodium iodide symporter). It is an active transport through an electrochemical gradient maintained mainly by adenosine triphosphatase enzyme. NIS is expressed in many other tissues, including salivary gland, mammary gland, and gastric mucosa [4]. This active transport mediated by NIS can be competitively inhibited by thiocyanate (SCN^-), perchlorate (ClO_4^-), pertechnetate ($^{99m}TcO_4^-$) and perrhenate (ReO_4^-) [5]. TSH (thyroid stimulating hormone) is the principal regulator of NIS expression through a cyclic adenosine monophosphate mechanism. So, thyroid iodide anion accumulation is stimulated by TSH [6]. The I^- is subsequently transported from the intracellular domain to apical membrane by a PDS gene product termed pendrin. It is a highly hydrophobic transmembrane protein with 11 putative transmembrane domains [7]. Pendrin is also expressed in other tissues, such as the kidney and the inner ear. Iodide can be generated also by iodotyrosine deiodinase that catalyses deiodation of intermediate products for thyroid hormones formation, such as mono-iodotyrosine and di-iodotyrosine with a greater rate for mono-iodotyrosine [8]. A decade ago, a putative new transporter involved in iodide transfer through the apical membrane of follicular cells was identified and termed human apical iodide transporter [9]. Continuously, iodide is released into the follicular lumen, which contains the colloid.

Iodide oxidation and organification occur mainly in the apical surface (colloid-facing) of thyroid cells and these reactions catalyzed by thyroperoxidase (TPO) in the presence of hydrogen peroxide leading to active thyroid hormones. Thyroid hormone synthesis began by oxidation of iodide and its incorporation into iodotyrosines previously cited, such as mono-iodotyrosine and di-iodotyrosine (organification). These precursors of thyroid hormones are incorporated into thyrglobulin considered as a pool of thyroid hormone: thyroxin (T4) and 3,5,3′ tri-iodothyronine (T3).

3. Status of iodine deficiency and global epidemiology

3.1. Epidemiological data

Available data about UI concentrations cover 97.8% of school-aged children in the world [10]. This data indicates that the proportion of school-aged children and in the general population suffering from ID has respectively decreased by 8.5% and 5.8% in Europe. Americas present the lowest proportion for school-aged children affected by ID that is about 13.7%. About 73.3 millions of the world school-aged children are still suffering from ID [11]. Details of countries having iodine deficiency or excess were cited in Table 2 [12].

National surveys for iodine status, based on the median UI concentration measure, have revealed that a significant progress was made toward the elimination of iodine deficiency. In fact, 45 countries have progressed to adequate iodine status from 2003 until 2012 [11]. The greatest proportion of individuals with insufficient iodine intake in general populations were for Africa (55%).

The prevalence of household consuming iodized salt was 74% for Asia (55% for south Asia and East Asia, 88% for The Pacific), 61% for the least developed countries and 71% for developing countries. This means the proportion over the whole world was 71% [13]. Until today, iodine intake is moderately