Chronic Lymphocytic Thyroiditis in a 5-Year-Old Girl

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UNTIL the last decade it was not recognized that chronic lymphocytic thyroiditis was a common cause of thyroid enlargement in children and adolescents. In 1954, Gribetz, Talbot and Crawford1 reviewed the literature and found only 11 reported cases, to which they added six of their own. However, two years later a surge of interest in Hashimoto's thyroiditis was initiated by the investigations reported by Witebsky and his co-workers2,3 and by Roitt et al.4 Before long many aspects of the etiology, immunology and pathogenesis of thyroiditis were being studied by workers who had been stimulated by the implications of the first reports. As a result, there now exists an impressive accumulation of data which support the concept that several common disorders of the thyroid, including chronic lymphocytic thyroiditis in its various forms and primary hypothyroidism, may represent autoimmune responses of the gland to antithyroid antibodies. By the use of appropriate techniques it is usually possible to demonstrate circulating antibodies, directed against one or more constituents of thyroid tissue, in the blood taken from patients with these disorders. Although of considerable diagnostic significance, it is doubtful whether such humoral antibodies have any direct pathogenic effect on the thyroid in vivo. Rather, it is believed that thyroid autodestructive lesions represent an immune response of the delayed type associated with cell-bound antibodies. Frequently a family history of thyroid disorders can be elicited from a patient with thyroiditis.5,6 This has led to the formulation of a hypothesis, that the formation of thyroid auto-antibodies may be the result of a genetically determined disorder of immunological tolerance.7

Since 1954, several investigators have reported a large number of new cases of juvenile lymphocytic thyroiditis. Undoubtedly these do not represent an actual increase in incidence of the disease (which some studies suggest may also be occurring) but rather reflect a greater awareness on the part of clinicians as a result of the increased interest in the immunologic aspects of thyroid function. Four large series totalling 128 subjects form the greatest proportion of the newly reported cases.5-12 As a result of these studies, it is now recognized that chronic lymphocytic thyroiditis as it occurs in childhood is probably related to adult Hashimoto's disease, but often does not share several of the clinical and pathological features of the classical disease as first described by Hashimoto.

The present report describes the case history and laboratory findings in a young girl with chronic lymphocytic thyroiditis diagnosed by tissue biopsy. Her case is of interest in that she demonstrates as an individual many of the features of the disease which have been reported to occur with varied incidence in any large series of young patients with thyroiditis. In addition, a number of aspects of iodine metabolism have been studied in this patient in order to compare them with those which have been reported in older age groups.5,13

Materials and Methods

Except for the serum protein-bound iodine (PBI) and butanol-extractable iodine (BEI) values for the 1965 admission, all protein-bound iodine determinations were performed at the University of Alberta Hospital.† Serum thyroxine was determined by the method of Murphy and Pattee14 as modified by Campbell,15 using albumin-coated charcoal.

Antibody to thyroglobulin was estimated by a modification of the tanned red cell hemagglutination technique of Boyden16 using preserved thyroglobulin-sensitized sheep cells.‡ Thyroid microsomal antibodies were determined by the complement-fixation method of Roitt and Doniach.17‡

Thyroid uptake of 131I was performed by the Radiosotope Laboratory of the University of Alberta Hospital by a routine method with oral administration of the tracer.

Twenty-four hours before the open thyroid biopsy the patient was given 400 µc. 131I so that the distribution of the tracer within the thyroid and serum could be determined by chromatography and differential ultracentrifugation. A portion (1.0 g.) of the thyroid tissue obtained at surgery was homogenized in 10 ml. isotonic

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†Determined by Bio-Science Laboratories, 12330 Santa Monica Blvd., Los Angeles 25, California.
‡Using a Technicon AutoAnalyzer.
†Burroughs Wellcome.
1.15% KCl containing 10 μmoles thiourea and separated into a nuclear, a mitochondrial, a microsomal and a soluble fraction by the method of DeGroot and Carvalho.18 The 131I activity of each fraction was determined and expressed as a percentage of the total. A second portion of the thyroid tissue was homogenized and subjected to hydrolysis with pronase.19 The incubation mixture was extracted with acid butanol, which was then neutralized with NH₄OH and evaporated to dryness. The residues were dissolved in a small volume of alkaline butanol and aliquots chromatographed by an ascending technique on strips of Whatman No. 1 filter paper using butanol: ethanol: 2N ammonia (5:1:2) as solvent system. Radioactive portions of the chromatograms were identified by radioautography. The 131I activity of each radioactive spot on the chromatogram was determined by counting the appropriate portion of the paper in a well-type scintillation counter. The results were expressed as a percentage of the total 131I activity of the chromatogram.

The butanol-soluble 131I components of serum were separated and identified in a similar manner using paper chromatography following acidification of the serum and extraction with butanol. In addition, aliquots of the thyroid supernatant fraction and of the blood serum were studied by electrophoresis on paper at 225 volts for 60 minutes in pH 8.6 barbital-citrate 0.1 M buffer. Radioautography was used to locate radioactive portions of the strips for quantitative measurements.

**Case Report**

Y.V., a 5½-year-old girl, was first seen at the University of Alberta Hospital in March 1965, because of a goitre which was increasing in size. She had been born at term, the second of three children. Her mother reported that she had been slightly slower in both mental and physical development than her older sister and younger brother, and had always been a very active, nervous child. She had a history of recurrent ear infections. At the time of admission to hospital the mother also commented on the patient’s small size, dry skin and troublesome constipation. She stated that a slight enlargement of the girl’s neck had been noted soon after her birth, but no noticeable change in the goitre occurred until after her fifth birthday. This rapid increase in the size of the patient’s neck during the three months before her admission to hospital prompted her mother to seek medical attention.

Although none of the patient’s immediate family (parents or siblings) had a history or signs of thyroid dysfunction, there was a strong history of goitre and thyrotoxicosis on both the maternal and paternal sides of the family. The patient’s parents were not related, being, in fact, of different nationalities.

On physical examination the patient was a small, restless child with a prominent enlargement of the neck (Fig. 1). Her skin was dry. On palpation the thyroid gland was estimated to be 40 to 50 g in size. It was diffusely enlarged and both the isthmus and pyramidal lobe were readily felt. The surface was lobulated. The gland moved freely and was non-tender. The only other significant physical findings were bilaterally scarred ear drums and a delay in the relaxation phase of the deep tendon reflexes at the ankles.

The patient’s physical and mental development were assessed (Table 1). Her dimensions were found to be below the 10 percentile limits and her bone age of 4½ was approximately 1 year behind her chronological age. The I.Q. assessment showed her to be of low to middle-average intelligence.

Laboratory findings at the time of admission are summarized in Table 1. Of particular interest was the large discrepancy of over 9 μg per 100 ml between the protein-bound iodine (PBI) and butanol-extractable iodine (BEI) values. The uptake of 131I by the thyroid gland following the oral administration of 10 μc. of the tracer is illustrated in Fig. 2. The maximum value of 24% was attained in two hours, suggesting that the gland was under high stimulation. This is compatible with the suggestion that circulating levels of pituitary thyroid-stimulat-
ing hormone (TSH) are increased in thyroiditis. The measurement of thyroidal uptake of $^{131}$I was repeated; four hours after a second drink of radiiodine, 400 mg. potassium perchlorate was given orally (Fig. 2). The fall in thyroidal $^{131}$I activity which occurred over the next few hours is characteristic of defective organic binding of iodine within the gland. However, since the effect of perchlorate was manifest over several hours, rather than being the result of a prompt and precipitous fall in thyroid $^{131}$I, it is probable that the observed decrease in radioactivity of the gland was a composite of several factors: (a) defective iodontation of tyrosine with an increased intrathyroidal pool of diffusible iodide; (b) defective coupling of iodothyrosines with rapid deiodination of $^{131}$I-iodotyrosine to iodide, and (c) an increased rate of secretion or diffusion of organic iodine into the blood serum from the gland. Rapid rates for the latter two processes would result if levels of circulating TSH were elevated. No increase in the 24-hour thyroidal uptake of $^{131}$I occurred after an intramuscular injection of TSH.

Circulating thyroid autoantibodies were measured on several occasions by two methods. Very low titres of antithyroglobulin antibodies were detected consistently by the tanned red-cell hemagglutination procedure. Similarly, small concentrations of antimicrosomal antibodies were found to be present in the serum by the complement-fixation method. The demonstration of antithyroid antibodies of low titre has been a common finding in reported cases of juvenile lymphocytic thyroiditis.

Other pertinent laboratory findings included a total serum protein of 8.2 g. per 100 ml., with albumin 4.0 g. and globulin 4.2 g. Electrophoresis of the serum proteins revealed that the concentration of $\gamma$-globulin was increased moderately at 2.2 g. per 100 ml. Serum cholesterol was 210 mg. per 100 ml. and thymol turbidity was abnormal at 15 units (normal: 0 to 4 units).

An open biopsy of the thyroid gland was performed 24 hours after the administration of 400 $\mu$g. $^{131}$I. At surgery, two portions of the greatly enlarged thyroid isthmus were removed. One portion was sectioned for microscopic studies; the other was utilized to determine the $^{131}$I distribution within the tissue. The histology of the gland (Fig. 3) revealed marked lymphocytic infiltration with forma-
TABLE II.—Significant Physical and Laboratory Findings at Time of Second Admission (1968), Age 8 Years, 6 Months

<table>
<thead>
<tr>
<th>Patient Y.V.</th>
<th>Normal median value or normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height</td>
<td>47.5&quot;</td>
</tr>
<tr>
<td>Weight</td>
<td>45.0 lbs.</td>
</tr>
<tr>
<td>Radiological bone age (wrist)</td>
<td>7.5 years</td>
</tr>
<tr>
<td>Thyroid gland</td>
<td>40-50 g.</td>
</tr>
<tr>
<td>Serum PBI*</td>
<td>5.4 µg./100 ml.</td>
</tr>
<tr>
<td>Serum thyroxine*</td>
<td>1.8 µg./100 ml.</td>
</tr>
<tr>
<td>PBI—Serum thyroxine*</td>
<td>3.6 µg./100 ml.</td>
</tr>
<tr>
<td>Thyroid uptake of 131I†</td>
<td>&lt;20 g.</td>
</tr>
<tr>
<td>4 hours</td>
<td>9.3%</td>
</tr>
<tr>
<td>24 hours</td>
<td>8.2%</td>
</tr>
<tr>
<td>Antithyroglobulin antibodies (TRC)</td>
<td>1/25</td>
</tr>
<tr>
<td>Antimicrosomal antibodies (CF).</td>
<td>+</td>
</tr>
</tbody>
</table>

*Patient receiving daily 1 grain desiccated thyroid. †Thyroid medication discontinued for one week.

A portion of the same thyroid tissue was homogenized, hydrolyzed with pronase and extracted with butanol. The radioactive components of the extract were separated and identified by paper chromatography and radioautography. The relative 131I-activity of each was determined (Table IV). Almost one-half of the total radioactivity of the butanol extract of the thyroid hydrolysate was composed of moniodotyrosine (MIT). The ratio of MIT to diiodothyrosine (DIT) was increased markedly above normal, being 1.7. The butanol-extractable radioactivity present in the blood serum was largely inorganic iodide. A trace of hormonal iodine was detected. Slightly larger amounts of radioactivity were accounted for by MIT and DIT.

In addition to the chromatographic studies, aliquots of both the blood serum and the soluble fraction of the thyroid homogenate from the same patient were subjected to electrophoresis on paper strips at pH 8.6 in barbital buffer.

TABLE IV.—Percentage Distribution of Butanol-Soluble 131I in Blood Serum and Thyroid Gland by Paper Chromatography

<table>
<thead>
<tr>
<th>Compound</th>
<th>Thyroid*</th>
<th>Serum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iodoprotein (origin)</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>Iodide</td>
<td>16</td>
<td>77</td>
</tr>
<tr>
<td>MIT</td>
<td>(1.0\times10^{5})</td>
<td>47</td>
</tr>
<tr>
<td>DIT</td>
<td>(1.0\times10^{4})</td>
<td>6</td>
</tr>
<tr>
<td>T3 + T4</td>
<td>28</td>
<td>4</td>
</tr>
<tr>
<td>MIT/DIT</td>
<td>6</td>
<td>2</td>
</tr>
</tbody>
</table>

*Homogenate subjected to hydrolysis with pronase.
Of the total $^{131}$I associated with the serum proteins, approximately 80\% was found to be insoluble in acid butanol. The butanol-insoluble iodoprotein, on electrophoresis, corresponded to serum albumin as reported by previous investigators.$^5$, $^{22}$, $^{23}$ A similar albumin-like iodoprotein could be demonstrated in the soluble fraction of the thyroid homogenates. The relatively large concentration of this abnormal iodoprotein in the circulation would explain the impressive difference between the values of serum PBI and BEI (or T4).

**Interpretation of Results**

DeGroot et al.$^5$ have shown that there is an abnormal distribution of $^{131}$I among the subcellular fractions of thyroid cells from glands of patients with Hashimoto's thyroiditis who have received the tracer 24 to 200 hours before operation. The alteration consisted of a relative increase in particulate radioactivity and a decrease in the soluble fraction. The change was characteristic not only of chronic lymphocytic thyroiditis, but also of any thyroid gland that was very active with a small store of iodinated thyroglobulin. A similar abnormality was found in the biopsy material from the present case.

Based on their extensive studies of iodine metabolism in Hashimoto's thyroiditis, Volpé et al.$^{13}$ have reported that a very common alteration which can be demonstrated in this disease is a decrease in thyroidal DIT relative to MIT production. Whereas the normal MIT/DIT ratio was 0.5 to 0.9, the range of results from their studies was 1.26 to infinity. All but one of the patients investigated were adults, the exception being a 13-year-old girl. In the present study involving a 5-year-old patient, a similar alteration in iodine metabolism was found. Volpé and his colleagues have also reported the abnormal finding of labelled MIT and DIT in the serum from most of their cases.

DeGroot and his colleagues$^5$ have proposed that the unusual iodoprotein found in the thyroid serum of many patients with thyroiditis may reflect a decreased thyroglobulin synthesis with iodination of an alternate protein formed as a result of the marked hyperplasia of the gland.

Because lymphocytic thyroiditis was suspected in all children with non-toxic goitre who presented to these clinics, the rate of diagnosis was found to be very much higher than predicted from the sporadic reports before that time. If the true incidence of the disease is reflected by that found by such centres, which employ a higher index of suspicion and more complete investigation than elsewhere, it must follow that many children and adolescents with lymphocytic thyroiditis still go undiagnosed.

In addition to emphasizing the relatively common occurrence of this disease, the reported large series have helped to delineate the criteria which are most likely to be useful to the clinician for the investigation, diagnosis and treatment of juvenile thyroiditis.

**Presentation**

In at least one-half of the cases the only symptom reported by the patients when first seen by the clinic was a goitre which had developed insidiously over a period of weeks, months or years. Less commonly the goitre was accompanied by symptoms (usually mild) of hypothyroidism or of local pressure in the neck. Very rarely the onset of the disease was subacute, associated with local pain and tenderness.$^{24}$

**Incidence**

This disease was much more common in females (90\% of reported cases) than in males. Thyroiditis has been reported to occur at all ages, even as young as 3 months,$^8$ but the diagnosis was uncommon in very young children. The incidence of diagnosis increased rapidly in children over 5 years of age, with a peak occurring at 11 to 12 years. Thereafter there was a gradual decline in incidence with age, so that it was uncommon to diagnose lymphocytic thyroiditis in the age group 20 to 30 years. After the fourth decade, the incidence of adult Hashimoto's disease has been found once again to rise markedly.$^{26}$ A positive family history of thyroid disorders was elicited in approximately one-half of the reported juvenile cases.

**Symptoms**

In children in whom symptoms other than goitre were reported, those most common were nervousness, irritability, fatigue, cold intolerance, hoarseness or dysphagia.$^8$ Although transient symptoms suggestive of minimal hyperactivity of the thyroid were not uncommon, overt hyperthyroidism was rare. In some series a high incidence of various non-thyroidal allergic manifestations was reported.$^8$
Signs

The enlarged thyroid gland associated with virtually all of the reported cases of juvenile lymphocytic thyroiditis was characteristically two to four times normal size, the involvement being diffuse, including the isthmus and frequently the pyramidal lobe. The goitre was usually reported as being moderately firm in consistence, mobile and non-tender. Frequently the surface of the gland was irregular or lobulated and finely granular. Less common were distinct nodules. Occasionally a brisk or thrill could be detected over the enlarged gland. Most children, when the disease was first diagnosed, were clinically euthyroid, but a significant proportion were found to be mildly hypothyroid. Severe myxedema was uncommon at the onset, but a distinct increase in incidence of hypothyroidism was found to occur with time when any group was followed up for a period of months or years. The majority of subjects were of normal height and weight at the time of diagnosis; those who fell below the lower limits of normal were almost always those in whom a clinical diagnosis of hypothyroidism was obvious.

Laboratory Findings

Because lymphocytic thyroiditis is a progressive process, the common laboratory tests of thyroid function gave an extremely variable pattern of results when applied to any group of patients with the disease. However, when the results were compared from subjects of the same age group and at the time when their thyroiditis was at approximately the same stage, a more uniform distribution was obtained.

At the time of diagnosis the most characteristic laboratory finding was a difference of more than 1.0 mcg per 100 ml. between the values of protein-bound iodine (PBI) and butanol-extractable iodine (BEI). At first the PBI was usually in the high or high-normal range, but as the disease progressed it was found to decrease. In a significant proportion of untreated subjects, the value was found eventually to fall within the hypothyroid range. It has been demonstrated that the discrepancy between the PBI and BEI values was related to the presence of a butanol-insoluble, metabolically inactive iodoprotein in the circulation. The same type of circulating iodoprotein, which has the electrophoretic mobility of albumin, has been reported in a variety of thyroid disorders including Graves’ disease, thyroid malignancy and one form of goitrous cretinism.

Also characteristic of the early stages of juvenile lymphocytic thyroiditis was a rapid, occasionally elevated thyroidal uptake of radioactive iodine (RAI). The uptake was usually insensitive to exogeneous TSH, but was readily depressed by the administration of thyroid hormone or iodide. It has been assumed that these laboratory findings and the hypertrophy of the gland are related to a high level of thyroid stimulation by pituitary thyrotropin (TSH), which was being secreted in response to a subnormal concentration of circulating, metabolically active thyroid hormone. However, few direct measurements of circulating TSH in juvenile thyroiditis actually have been performed by the newer, more sensitive assay procedures. Until these are reported, the role of TSH in the pathogenesis of the goitre and altered iodine metabolism of thyroiditis must remain speculative.

It has been found in approximately one-half of the subjects investigated that a defect in the thyroidal incorporation of iodine into organic form can be demonstrated by administering perchlorate in conjunction with the RAI uptake test. In such cases perchlorate was found to cause a release from the gland of more than 5% of the accumulated RAI.

Although total serum protein levels of patients with lymphocytic thyroiditis were usually normal, frequently an elevation of serum \( \gamma \)-globulin could be demonstrated. Such increases may give rise to the rather common abnormal flocculation tests which have been reported in these cases.

The association of circulating antithyroid antibodies, often of high titre, with Hashimoto’s thyroiditis has been extensively documented. A variety of procedures may be used successfully to demonstrate the different abnormal auto-antibodies that have been described (Table V). In their large series of patients with juvenile lymphocytic thyroiditis, Doniach, Nilsson and Roitt found that when more than one method for detecting the antithyroid antibodies was utilized, positive results could be obtained in virtually every case. However, as a group, children did not give the markedly elevated titres commonly found in adult Hashimoto’s disease. It was not uncommon to find low or negative titres in proved cases of the disease when only one method was chosen. Euthyroid, non-goitrous relatives of the patients have been shown to have circulating antithyroid antibodies with a significantly greater frequency than that occurring in the general population. Furthermore, it has been reported that the serum of the patients themselves may contain, with significant frequency, non-thyroidal autoimmune antibodies in addition to those directed against thyroid.
TABLE V.—CIRCULATING ANTITHYROID ANTIBODIES IN LYTIC PHCYTIC THYROIDITIS

<table>
<thead>
<tr>
<th>Antigen</th>
<th>Procedure for antibody detection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thyroglobulin</td>
<td>(1) Precipitin</td>
</tr>
<tr>
<td></td>
<td>(2) Tanned red cell hemagglutination</td>
</tr>
<tr>
<td></td>
<td>(3) Latex particle agglutination</td>
</tr>
<tr>
<td></td>
<td>(4) Coons fluorescent staining (fixed thyroid sections)</td>
</tr>
<tr>
<td>Microsomal (intracellular)</td>
<td>(1) Complement fixation</td>
</tr>
<tr>
<td></td>
<td>(2) Coons fluorescent staining (unfixed thyroid sections)</td>
</tr>
<tr>
<td></td>
<td>(3) Demonstration of cytoxic activity using thyroid-tissue culture</td>
</tr>
<tr>
<td>Second colloid component (not thyroglobulin)</td>
<td>(1) Coons fluorescent staining (fixed thyroid sections following adsorption of serum with thyroglobulin)</td>
</tr>
</tbody>
</table>

with the theory which proposes that the basic defect common to all autoimmune disease is a genetically determined disorder of immunological tolerance.7

Histology

In spite of the better understanding of the clinical and laboratory features of juvenile lymphocytic thyroiditis that has resulted from the recently reported cases of the disease, a firm diagnosis still can only be made from the direct histological examination of thyroid tissue. Many centres report that suitable tissue for pathological studies may be obtained with a high frequency of success and a low incidence of complications by needle biopsy. In such centres it has become uncommon to perform open biopsy unless surgery is indicated for compression symptoms or suspected malignancy. On microscopic section, the thyroid of juvenile thyroiditis characteristically has been found to be diffusely infiltrated with lymphocytes and plasma cells between the follicles. Frequently, focal collections of lymphocytes with the formation of germinal centres were reported. The follicles themselves were found to be atrophied with evidence of epithelial degeneration and they contained little or no colloid. Classical oxyphilic changes in the epithelial cells were observed only rarely and fibrosis was not a prominent feature. The histological features found in the childhood diseases were quite different from the classical changes first reported by Hashimoto26 and which seem to occur almost exclusively in adults. Juvenile lymphocytic thyroiditis may represent a separate disease or, more likely, an earlier stage of the same disorder seen in adults. The reported results from the few cases in which serial biopsies have been taken would support the latter concept.

Therapy

With very few exceptions, for children and adolescents with juvenile lymphocytic thyroiditis thyroid hormone has been prescribed. Certainly there is no question that any patients who are hypothyroid at the time of diagnosis must be treated in this manner. Furthermore, since the disease is of a progressive nature, the chance that a subject will develop hypothyroidism increases with time. Beginning replacement therapy with thyroid hormone when the diagnosis is first made, even in euthyroid subjects, will prevent the possible occurrence of unrecognized (and therefore untreated) periods of hypothyroidism. A second reason for treating these children with thyroid hormone has been the attempt to reduce the degree of thyroid enlargement, presumably by inhibiting excessive stimulation of the gland by pituitary TSH. In this regard, the reported results have been most variable. In any large series a small number of treated patients respond with a dramatic disappearance of their goitre. In another small group, progressive thyroid enlargement is found. The remainder, which make up the majority of cases, have minimal to moderate changes in goitre size, approximately 50 to 75% of the subjects experiencing a slow but appreciable decrease in thyroid enlargement.

Summary

A case report has been presented describing chronic lymphocytic thyroiditis, diagnosed by an open biopsy of the thyroid, in a 5-year-old girl. This patient was found to exhibit the following physical and laboratory features of the disease, all of which have been described with variable incidence in older children and in adults, including the abnormalities in iodine metabolism: (1) a progressive, insidious, painless enlargement of the neck accompanied by mild symptoms of hypothyroidism, the thyroid gland becoming diffusely enlarged and firm; (2) a positive family history of thyroid disease; (3) a large discrepancy between the values for serum protein-bound iodine and butanol-extractable iodine, accompanied by the presence in the thyroid and serum of an abnormal, albuminlike, butanol-insoluble protein; (4) a rapid uptake of 131I which was unresponsive to exogenous thyrotropin but was sensitive to orally administered perchlorate; (5) the demonstration of circulating antithyroglobulin and antimicrosomal antithyroid antibodies of low titre; (6) an abnormal distribution of 131I among thyroid cell-fractions and iodinated compounds in thyroid tissue obtained at biopsy, with a markedly increased concentration of moniodotyrosine relative to diiodotyrosine.

This patient was treated with desiccated thyroid for three years, at the end of which time her thyroid status was reassessed. Most of the abnormalities found at the time of diagnosis were still demonstrable.
A brief review has been made of the common features of previously reported cases of juvenile lymphocytic thyroiditis for comparison with the findings in the present study.

Résumé Les auteurs présentent un cas de strumite lymphomateuse chronique, diagnostiquée par biopsie ouverte de la thyroïde, survenue chez une fillette de 5 ans. Chez cette malade, on notait diverses observations cliniques et certaines valeurs de laboratoire qui toutes avaient déjà été décrites, avec une fréquence variable, chez des enfants plus âgés et chez des adultes, notamment les anomalies frappant le métabolisme de l'iode: (1) tuméfaction progressive, insidieuse, indolore du cou, marquée par des symptômes frustes d'hypothyroidie; (2) des antécédents familiaux d'affection thyroïdienne; (3) une forte divergence entre les valeurs de l'iode protéique du sérum et celles de l'iode protéique par extraction butylique, s'accompagnant de la présence dans la thyroïde et dans le sérum d'une protéine albuminoïde, insoluble dans le butanol; (4) une fixation rapide de $^{131}$I par la thyroïde, laquelle ne réagissait pas à l'administration exogène de thyrotrophine, mais qui était sensible à l'administration per os de perchlorate; (5) la découverte d'une anti-thyroglobuline circulante et d'anticorps anti-thyroïdiens microsomiques de titre faible; (6) une distribution anormale de $^{131}$I dans les diverses fractions de la cellule thyroïdienne et dans les composés iodés du tissu thyroïdien obtenus par biopsie, notamment une augmentation nette de mono-iodotyrosine par rapport à la di-iodotyrosine.

La malade a été traitée au moyen de thyroïde desséchée pendant trois ans, l'état de sa thyroïde ayant été réévalué à la fin de cette période. La plupart des anomalies constatées au moment du diagnostic initial était encore présente.

Les auteurs passent brièvement en revue les caractéristiques courantes des cas de strumite lymphomateuse juvénile antérieurement rapportés, pour permettre une comparaison avec les observations de la présente étude.

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