status of the specialty has varied little over the years, and the existing variation is influenced in part by variations in the number of positions offered. The ranking according to the Autonomous Communities and centers shows important variations in the median order number, suggesting that certain predilections are relatively constant. The data presented correspond to an objective analysis of the choice of Endocrinology and Nutrition based on the official information of the MSSSI. Our study does not propose to provide an analysis of the care or teaching quality of the different resident training centers as this aspect has been addressed in other studies.[3,4] The choice of medical specialty and resident training center is influenced by a number of personal factors such as perception of the specialty, prestige, geographical preferences, etc.,[5–7] that have not been evaluated in this study. Nevertheless, this simple analysis presents data regarding the general level of interest in the specialty, and depicts the geographical and resident training center preferences of future residents.

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Influence of iodine status on maternal thyroid function during pregnancy☆

Influencia del estado de yodación sobre la función tiroidea materna durante la gestación

Iodine deficiency is an important public health concern and much time and effort has been spent in seeking to eradicate the problem over the last 80 years.[1] Pregnant women are particularly sensitive in this regard, due to their increased iodine requirements during pregnancy. The World Health Organization (WHO) recommends a daily iodine intake of 250 μg. A median urine excretion (ioduria) of 150–249 μg/l is indicative of adequate intake, while <150 μg/l is considered insufficient, 250–499 μg/l is above what the body needs, and >500 μg/l is considered excessive.[2]

Iodine nutritional status in the Spanish population is currently adequate.[3] In contrast to iodine deficiency, excesses from dietetic sources are infrequent here. However, the generalized use of iodized pharmacological supplements in pregnant women, together with the largely uncontrolled increase in the iodine contents of dietetic sources (silent iodoprophylaxis), and other potential contributing factors such as the use of iodized antiseptics, can result in excessive intake – with consequent but less well-known repercussions upon maternal thyroid gland function.[4]

A study was made of 106 healthy pregnant women with normal thyroid function and negative thyroid immune findings. We determined the levels of TSH, FT3 and FT4 in the first (week 10–12) and third trimester (week 34–36) with a chemiluminescence microparticle immunoassay in an ARCHITEC analyzer (Abbott Ireland Diagnosis Division, Linamuck, Longford, Ireland). In the case of TSH the sensitivity of the assay is ≤0.01 μIU/ml, and the normality value (NV) ranges from 0.49 to 4.67 μIU/ml; the specificity of the assay is <10% cross-reactivity with TSH, FSH and hCG. In the case of FT4, the limit of detection is ≤0.4 ng/dl, and the NV is

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Table 1 Thyroid function parameters according to ioduria level.

Ioduria (μg/l)	n (%)	First trimester			n (%)	Third trimester		
		TSH μIU/ml	FT4 ng/dl	FT3 pg/ml		TSH μIU/ml	FT4 ng/dl	FT3 pg/ml
<150	50 (47.1)	1.88 ± 1.06	1.06 ± 0.09	2.97 ± 0.36	39 (36.7)	1.96 ± 0.76	0.87 ± 0.08	2.95 ± 0.34
150–249	20 (18.8)	1.43 ± 0.50	1.07 ± 0.07	2.94 ± 0.26	25 (23.5)	1.95 ± 0.70	0.89 ± 0.09	2.92 ± 0.35
≥250	36 (33.9)	1.67 ± 1.04	1.07 ± 0.09	2.93 ± 0.33	42 (39.6)	1.75 ± 0.70	0.88 ± 0.10	2.87 ± 0.29
p-Value		0.303	0.966	0.869		0.248	0.790	0.556

Data reported as the mean ± standard deviation.
$p < 0.05$ was considered significant.

0.70–1.59 ng/dl; the specificity of the assay is ≤0.0035% cross-reactivity with T3. In the case of FT3, the limit of detection is ≤1 pg/dl, and the NV is 1.71–3.71 pg/dl; the specificity of the assay is <0.001% cross-reactivity with T4. In addition, we collected a sample of first morning urine to determine ioduria using high performance liquid chromatography (Immunochrom GMbh, Heppenheim, Germany). The intra- and inter-assay coefficient of variation was <3.5% and <4.8%, respectively, with a sensitivity of 0.02 μmol/l.

The mean age of the pregnant women was 31.5 ± 4.7 years, with a body mass index (BMI) of 24.4 ± 5.0 kg/m^2. There were 25 smokers (23.5%). Forty-two women were in their first pregnancy (39.6%), and 26 had a history of at least one aborted pregnancy (24.5%). Iodized salt was consumed by 42 patients (39.6%) in the first trimester and by 100 (94.3%) in the last trimester ($p < 0.05$). Pharmacological supplements were being used by 79 patients (74.5%) in the first trimester and by 102 (96.2%) in the last trimester ($p < 0.05$).

Median ioduria in the first trimester was 171.31 μg/l (interquartile range [IQR] 184.18) versus 190.37 μg/l (IQR 263.56) in the third (p = ns). Only 20 pregnant women in the first trimester (18.8%) and 25 in the third trimester (23.5%) showed optimum ioduria levels (defined as 150–249 μg/l). However, a decrease was observed in the number of women with ioduria <150 μg/l, from 50 in the first trimester (47.1%) to 39 in the third (37.7%). By contrast, the number of pregnant women with ioduria ≥250 μg/l increased from 36 in the first trimester (33.9%) to 42 in the third (39.6%).

Table 1 shows the relationship between ioduria and the different thyroid function parameters in both the first and the second trimester of pregnancy. Although the women with adequate ioduria (150–249 μg/l) presented lower TSH levels in the first trimester, the difference was not significant. No differences were observed in the FT3 or FT4 levels between the groups.

Only 39.6% of the pregnant women in this study consumed iodized salt in the first trimester. This percentage was far below the level considered necessary to ensure an adequate dietetic supply, and it was particularly worrying given that prolonged consumption for at least two years is required in order to reduce the risk of thyroid gland dysfunction during pregnancy.[5] The significant increase in consumption observed following the recommendation reflects the importance of the promotional measures taken.

Iodized supplements were predominantly used. However, only 11 of the 79 pregnant women who used these supplements in the first trimester started to use them at least 8 weeks before admission to the study. These women presented lower mean TSH values than those who started taking the supplements later (1.22 μIU/ml ± 0.6 vs 1.78 μIU/ml ± 1.0), though the difference was not significant ($p = 0.13$). This effect has been previously described and was attributed to a stunning effect on the thyroid gland secondary to a sudden rise in iodine levels.[6]

Thyroid function problems related to iodine deficiency are well known.[7] In the present study, ioduria <150 μg/l did not result in significant changes in any of the thyroid function parameters versus the pregnant women with ioduria 150–249 μg/l. However, only 7 women (6.6%) in the first trimester and 5 in the third (4.7%) presented ioduria <50 μg/l, these being the patients with the highest risk of thyroid gland dysfunction.[8]

Iodine excess can also affect thyroid function.[9] The pregnant women in our study with ioduria ≥250 μg/l showed no differences in thyroid function parameters versus the women with ioduria values within the optimum range. In a previous study, pregnant women with a median ioduria >1000 μg/l showed higher TSH levels and a greater prevalence of subclinical hypothyroidism.[10] However, in our study only two women in the third trimester of pregnancy versus none in the first trimester presented ioduria >1000 μg/l.

As the main limitation of our study, mention must be made of the great intra- as well as inter-individual variability of ioduria, which can cause classification errors when the sample is being divided into sub-groups.

In conclusion, the pregnant women of this study showed adequate iodine status. The use of iodized pharmacological supplements was a predominant practice from the early stages of pregnancy, though not so the consumption of iodized salt. Maternal thyroid function showed no significant differences in the different iodine ranges analyzed.

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Autonomic enteropathy: A frequently ignored diabetic complication

Enteropatía diabética: una complicación frecuentemente ignorada

Gastrointestinal disorders are common in the general population and even more frequent in diabetics.[1] More specifically, high volume watery diarrhea may have a complex and multifactorial etiology, run a chronic and relapsing course and be refractory to conventional antidiarrheal treatments.[2] There are no specific recommendations for a diagnostic approach. We report a patient with high volume diarrhea, refractory to several empirical therapies. He required hospital admission due to dehydration and electrolyte imbalance, during which he underwent diagnostic procedures that allowed for the exclusion of small intestine and pancreatic disease and bacterial overgrowth. The diarrhea was attributed to diabetic enteropathy and there was a favorable response to clonidine.

An 81-year-old male was admitted in an Internal Medicine ward in July 2016 with severe and explosive watery diarrhea of twelve weeks duration associated to hypokalemia and dehydration. Bowel frequency was up to eight times per day and one to three times at night and was associated with flatulence, abdominal cramps, fecal urgency and a 13% weight loss since onset was documented. There was no reference to blood loss, fever, abdominal distension or other complaints. Over the past 3 months he had been admitted on two previous occasions with similar episodes and had been treated unsuccessfully with fasting, hydration, ciprofloxacin, metronidazole, probiotics and loperamide. There was no epidemiologic association and no surgical history.

Type 2 diabetes mellitus had been diagnosed 30 years prior to the current admission with established macro and micro-vascular complications such as retinopathy, nephropathy and diabetic foot and peripheral neuropathy. He also suffered from arterial hypertension, New York Heart Association class II heart failure and anemia of chronic disease. Current medication included lispro-insulin (10 U-morning, 8 U-night), linagliptin 5 mg od, isossorbid-dinitrate 20 mg od, lercanidipin 10 mg od, furosemide 40 mg od, rosuvastatin 10 mg od, lansoprazol 30 mg od and pregabalin 75 mg bd. Potassium-chloride 600 mg od had been added from the previous hospital admission.

Physical examination revealed pallor, dehydration, blood pressure 162/97 mmHg and heart rate 71 beats/minute. The abdomen was tender but without peritoneal irritation and there was no organomegaly. The rest of the examination was unremarkable.

Extensive investigations failed to reach a specific diagnosis (Table 1). The absence of pancreatic calcifications and normal fecal fat excretion excluded chronic pancreatitis.

We decided to start a trial therapy with clonidine in a maximum dose of 0.3 mg td with a good clinical response (decreased stool weight and frequency and more consistency). We progressively reduced its doses to 0.15 mg bd with sustained clinical response. Nevertheless some side effects were noted, namely: dry mouth, drowsiness (both disappeared after 48 h) and asymptomatic hypotension that was controlled with the reducing doses of clonidine and the anti-hypertensive drugs.