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Association between Helicobacter Pylori infection and autoimmune hypothyroidism in Egyptian population

Fatma Kamel Hammad [1], Zeinab Abd El-Baset Hassan [1], Dina Mohammed Abaza [1],Sabila Gomma 'Mosua,[2] Ahmed Ahmed Saad [3] and Hesham Mohamed Abou El-Soud [1]
Endocrinology and Metabolism Department [1], Internal Medicine Department [2] (Al Azhar University for girls), Internal Medicine Department [3](Faculty of Medicine – Cairo University).

Abstract

Autoimmune hypothyroidism commonly affecting females is one of the commonest causes of thyroid disease in adults. Among the various autoantibody tests applied in research and clinical practice, the determination of thyroid microsomal antibodies (TPO) and thyroglobulin antibodies (TG Ab) still retains its strong value in the screening for thyroid autoimmunity. Helicobacter pylori (H. pylori) infection plays an important role in the pathogenesis of chronic gastritis, peptic ulcer disease, MALT (Mucosa Associated lymphocyte T) Lymphoma and gastric cancer.

Aim of the work:

The aim of this work was to study the relationship between H.pylori infection and autoimmune hypothyroidism in Egyptian population.

Subjects and Methods:

This study was carried out on 147 Egyptian persons divided into 3 groups: **Hypothyroid Group:** Included 49 patients with autoimmune hypothyroidsm and positive antithyroid antibodies with no history of dyspeptic symptoms or peptic ulcer. **H.pylori positive Group:** Included 50 patients with dyspeptic symptoms or peptic ulcer with H.pylori positive antibodies with no history of any thyroid disease. **Control Group:** Included 48 apparently healthy persons serving as control. Serum Free T3, Free T4 and TSH were done for all subjects together with Antimicrosomal antibodies (TPO-Ab), Antithyroglobulin antibodies (TG-Ab) and Helicobacter Pylori antibodies (H. pylori Ab).

Result

There was no significant difference between all groups as regards age. Also there was significant difference between **Hypothyroid** and **H.pylori positive** groups as regarding TSH and Free T3, TG-Ab, TPO-Ab and H. pylori Ab. There is also significant difference between **Hypothyroid and control** groups regarding TSH, free T3, TG-Ab, TPO-Ab, and H. pylori Ab. There is significant difference between **H.pylori positive** and **control** groups regarding FT3 and H. pylori AB. **Hypothyroid Group** was divided according to the presence of H. pylori Ab into –ve &+ve H. pylori Ab subgroups. There was significant difference between the –ve and +ve subgroups as regard TSH, free T4 and TG-Ab. **H.pylori positive**

Group was divided according to the presence of TG Ab & TPO Ab into–ve and +ve subgroups. There was significant difference between the –ve and +ve cases in TSH, free T4, Free T3, and H.Pylori Antibody.

Positive correlation was found between H pylori Ab titer and age, TSH, TG-Ab and TPO-Ab titers. There was also negative correlation between H. pylori Ab titer and free T4. There is no correlation between H. pylori Ab titer and free T3. (Correlation is referred to all subjects of the study = 147).

Conclusion

This study revealed that patients with positive TG & TPO antibodies, showed (+ve) H. pylori Ab, with significant high titer in their sera. The patients with positive H. Pylori Ab showed high serum titer of TG-Ab. In our study H. pylori-Ab correlates to thyroid function tests and thyroid antibodies.
Key words: Thyroid peroxidase, Thyroglobulin, Helicobacter pylori, antibody.

Introduction

Hashimoto's thyroiditis (Ht) is the most common cause of thyroid disease in children and adolescents. It is also the most common cause of acquired hypothyroidism with or without goiter. The linkage between Ht and some HLA genes has been reported and genetic predisposition to thyroid autoimmunity is suggested by observation in twins (Lourini et al., 2003).Among the various autoantibody tests applied in reach and clinical practice, the determination of thyroid microsomal antibodies (TPO Ab) and thyroglobulin antibodies (TG Ab) still retains its strong value in the screening for thyroid autoimmunity (Scherbaum, 1987).Helicobacter pylori (H. pylori) infection plays an important role in the pathogenesis of chronic gastritis, peptic ulcer disease, MALT (Mucosa associated lymphocyte T) lymphoma and gastric cancer (Pellicano et al., 2001).On the basis of the levels of serum pepsinogen I (S-PGI) and gastrin-17 (SG-17) as well as H. pylori antibodies, it is possible to establish with high sensitivity and specificity whether the patient has gastritis, whether the gastritis is atrophic or not, and in which part of the stomach the atrophic changes are located (Sipponen et al., 2003).Higher serological prevalence rates of H.pylori infection have been reported in patients with autoimmune thyroiditis and its has been suggested that monoclonal antibodies against Cag-A positive H. pylori strains can cross react with follicular cells of the thyroid gland (Tomasi et al., 2005).Patients with autoimmune atrophic thyroiditis (AAT) have a markedly increased

prevalence of H pylori infection. Moreover, levels of anti-H.pylori IgG in AAT showed a strong relationship with the titers of microsomal autoantibodies. Nevertheless, as H. pylori infection increased the levels of gastrin, ppg I, and ppg II in infected patients with autoimmune thyroid disease (ATD), demonstrating significant gastric involvement, it is suggest that patients with AAT should be screened also for H pylori infection, because they may benefit from H. pylori eradication (Daniela et al., 2008).

The aim of this work was to:

Study the relationship between H.pylori infection and autoimmune hypothyroidism in Egyptian population.

Subjects and Methods:

Subjects

The present study has been conducted on 147 Egyptian subjects selected from the outpatient Endocrine and Metabolism clinic in EL Zahra Hospital. Their ages ranged from twenty (20) to fifty (50) years. They were divided into 3 groups as follows:

Hypothyroid group: included 49 patients (13 males and 36 females). They were chosen with evidence of clinical and biochemical hypothyroidism including positive antithyroid antibody .They has no history of any dyspeptic symptoms or peptic ulcer diseases.

H.pylori group II: included 50 patients, (13 males and 37 females). They had dyspeptic symptoms e.g., dysphagia, heart burn, regurgitation, retrosternal pain, nausea, vomiting, early satiety and sensation of distension. They had H. pylori positive antibodies with no history of any thyroid diseases. **Control group:** included 48 control subjects, (17 males and 31) females. They were apparently healthy, with no history of thyroid dysfunction, no history of dyspeptic symptoms.

All subjects were subjected to full history taking, complete clinical examination and routine laboratory investigations (Complete blood picture, fasting blood glucose level, liver enzymes and renal function tests).

Exclusion criteria: Diabetes mellitus, chronic liver diseases, malignancy and any patient suffering from any autoimmune diseases other than autoimmune thyroid diseases.

Methods

Laboratory Investigations:

All subjects in this study were subjected to the following:

1-Blood specimens were obtained and the usual precautions in the collection of venipuncture samples were followed strictly.

2- For accurate comparison to established normal values, fasting morning serum samples were obtained.

3-The blood was collected in a plain redtop venipuncture tube without additives

or anti-coagulants.

4- Blood was allowed to clot for serum sample.

5- Specimens were centrifuged to separate the

serum or plasma from the cells. Samples were refrigerated and stored at temperatures of -20ºC.

6-Thyroid function test:

a-Free T3 was measured using AccuBind ELISA Microwells Free Triiodothyronine (Monobind Inc., USA. Product code 1325-300).Reference range 3.1-6.8 pmol\l. Conversion factors: pmol/L x 0.651 = pg/ml. Free T4 was measured using AccuBind ELISA Microwells Free Thyroxin (Monobind Inc., USA. Product code 1225-300).Reference range 12-22 pmol\l. Conversion factors: pmol/L x 0.077688 = ng/dl. TSH was measured using AccuBind ELISA Microwells Free Thyrotropin (Monobind Inc., USA. Product code 325-300). Reference range 0.27-4.2 µIU\ml.TG-Ab was measured using AccuBind ELISA Microwells Free Thyroglobulin (Monobind Inc., USA. Product code 1025-300). Reference range up to 40 IU\ml. TPO-Ab was measured using AccuBind ELISA Microwells Free Thyroid peroxidase (Monobind Inc., USA. Product code 1125-300). Reference range up to 34 IU\ml Antibodies against H pylori IgG were determined using ELISA kits. Results were considered positive when higher than 20 U/ml. (Segni et al., 2004.).

Statistical analysis

Data of tests were analyzed using the arithmetic mean, standard deviation (SD), standard error (SE), unpaired students t- test.

The data were then analyzed statistically using SPSS statistical package version (12).

P> 0.05 insignificant(NS)

P < 0.05 significant (Sig.)

P < 0.01 highly significant (H.S)

Results

The summary data for laboratory investigations among studied groups are shown in (Table 1) .There was no significant difference between all groups as regard age. There was significant difference between hypothyroid and H pylori positive groups regarding TSH, Free T3, TG-Ab, TPO-Ab, and H.pylori Ab. There was also significant difference between hypothyroid and control groups regarding TSH, free T3, TG-Ab, TPO-Ab and H. pylori Ab. Also there was significant difference between hypothyroid and H pylori positive groups regarding FT3 and H pylori Ab. As regard (Table 2) There was significant difference between the –ve and +ve subgroups (regarding H pylori Ab) of hypothyroid group in TSH, free T4 and TG-Ab. While in (Table 3) there was significant difference between the –ve and +ve subgroups of H pylori positive group (regarding TG-Ab) for TSH, free T4, Free T3 and TPO- Ab, and H pylori-Ab. In (Table 4) correlations were done between H pylori Ab titre and all other studied parameters in all cases, there was significantly positive correlation between H. pylori Ab titer and

age, TSH, TG-Ab titer and TPO-Ab titer. There was also negative correlation between H. pylori Ab titer and free T4, but no correlation between H. pylori Ab titer and free T3 was found. Fig(1) shows the prevalence of males and females among both studied groups.

Table (1): Comparison between all studied groups as regard age, thyroid functions thyroid antibodies and H pylori antibody.

	Hypothyroid group Mean ±SD **(n=49)**	**H pylori positive group** Mean ±SD **(n=50)**	**Control Group I** Mean ±SD **(n=48)**	**P Value**
Age (years)	37.2±8.38	36.7±8.25	35.79±8.46	P1 : > 0.05 P2 : > 0.05 P3 : > 0.05
TSH(uIU\ml)	38.86± 2.89	5.63± 1.44	7.86± 1.86	P1 : <0.001 P2 : < 0.001 P3 : > 0.05
FREE T4(ng\dl)	0.51± 0.24	1.13±0.6	1.51±0.22	P1 : > 0.05 P2 : > 0.05 P3 : > 0.05
FREE T3(pg\ml)	2.75± 1.29	2.54± 0.67	2.88± 0.19	P1 : < 0.001 P2 : <0.001 P3 : < 0.05
TG-Ab titer(IU\ml	69.91±17.81	16.1±1.73	17.17±1.96	P1 : < 0.001 P2 : < 0.001 P3 : > 0.05
TPO-Ab titer(IU\ml)	39.24±1.7	6.66±1.35	9.16±1.6	P1 : < 0.001 P2 : < 0.001 P3 : > 0.05
H. pylori-Ab(IU\ml)	44.12±2.94	33.54±1.97	22.55±2.51	P1 : <0.05 P2 : <0.001 P3 : <0.05

P1: comparison between Hypothyroid & H pylori positive groups

P2: comparison between Hypothyroid &control groups

P3: comparison between H pylori positive & control groups

Table (2): Comparison between –ve and +ve subgroup (regarding H pylori Ab) in Hypothyroid group as regard age, thyroid function and thyroid antibodies.

		Number(49)	Mean ±SD	P
Age	-ve	10	33.1±9.01	> 0.05
(yrs)	+ve	39	38.26±7.99	
TSH	-ve	10	12.68±2.40	< 0.001
(ulU/ml)	+ve	39	45.57±2.87	
Free T4	-ve	10	0.72±0.15	< 0.001
(ng/dl)	+ve	39	0.46±0.22	
Free T3	-ve	10	1.17±0.50	> 0.05
(pg/ml)	+ve	39	3.15±1.45	
TG-Ab	-ve	10	48.57±10.23	< 0.001
(IU/ml)	+ve	39	75.38±15.00	
TPO-Ab	-ve	10	38.31±13.76	> 0.05
(IU/ml)	+ve	39	39.47±17.90	

Table (3): Comparison between –ve and +ve subgroup (regarding TG- Ab) in H lpylori positive group as regard age, thyroid function, TPO-Ab and pylori Ab).

		Number(50)	Mean ±SD	p
Age	-ve	45	36.42±8.44	>0.05
(yrs)	+ve	5	39.20±6.41	
TSH	-ve	45	2.55±1.79	<0.001
(ulU/ml)	+ve	5	33.30±3.78	
Free T4	-ve	45	1.20±0.26	<0.001
(ng/dl)	+ve	5	0.52±0.30	
Free T3	-ve	45	2.68±0.54	<0.001
(pg/ml)	+ve	5	1.33±0.59	
TPO-AB	-ve	45	4.70±3.93	<0.001
(IU/ml)	+ve	5	24.28±5.91	
Anti H. pylori	-ve	45	30.80±6.35	<0.001
(IU/ml)	+ve	5	58.14±5.84	

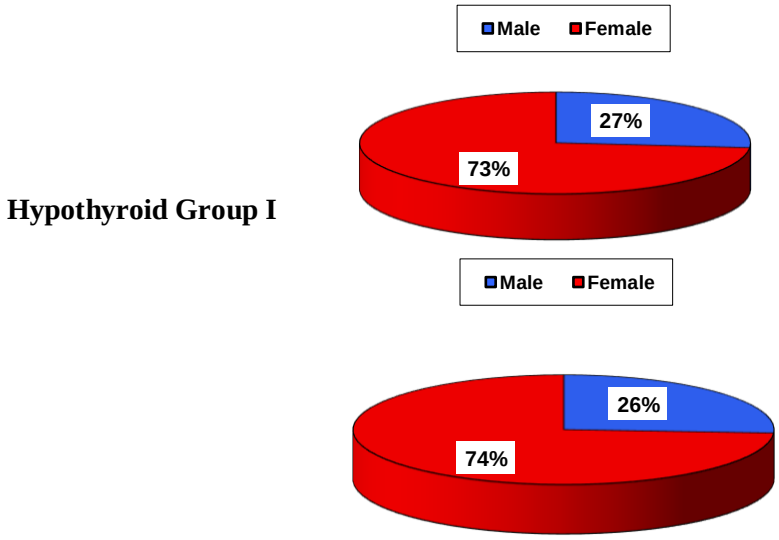
Table (4): **Comparison between –ve and +ve subgroup (regarding TPO- Ab) in H pylori positive group group as regard age, thyroid function, TG-Ab and pylori Ab).**

		Number(50)	Mean±SD	p
Age	-ve	43	36.6±8.35	> 0.05
(yrs)	+ve	7	37.2±8.17	
TSH	-ve	43	2.44±1.68	<0.001
(ulU/ml)	+ve	7	25.17±3.84	
Free T4	-ve	43	1.20±0.26	<0.001
(ng/dl)	+ve	7	0.65±0.33	
Free T3	-ve	43	2.70±0.53	<0.001
(pg/ml)	+ve	7	1.57±0.71	
TG-AB	-ve	43	10.9±6.40	<0.001
(IU/ml)	+ve	7	47.8±8.50	
Anti H. pylori	-ve	43	29.60±14.94	<0.001
(IU/ml)	+ve	7	57.73±24.71	

Table (**5**)**: Correlation between H pylori Ab titer and all other studied parameters in all cases (n=147).**

	r	p
Age	0.312	0.000
TSH	0.693	0.000
Free T4	-0.204	< 0.05
Free T3	-0.128*	> 0.05
TG-AB(titer)	0.629	0.000
TPO-AB (titre)	0.499	0.000

Figure (1): Distribution of cases according to sex among hypothyroid &H pylori positive groups



Discussion

Autoimmune hypothyroidism is a common disease that affects 1% of women but no more than 0.1% of men, generally between 40 and 60 years of age. As with other autoimmune diseases, susceptibility to autoimmune hypothyroidism is thought to be influenced by an interaction of genes and environment in addition to endogenous factors such as age and pregnancy (Austen et al., 2001).

Helicobacter pylori (H. pylori) infection is a chronic gastric gram negative infection that increases with age (Sallesan , 2007). This infection may induce a whole cascade of events leading to gastric pathologies such as peptic ulcer diseases, gastric precancerous lesions, and gastric lymphomas (Rieder et al., 2005). Moreover, an ongoing controversial role of this bacterium infection has been suggested in the etiopathogenesis of some extradigestive disease such as coronary artery disease, thrombotic disorders, primary biliary cirrhosis and Alzheimer's disease (Bernardini et al., 2007).

In the present study as regard age there was no significant difference between groups ($P<0.05$ for all), the subjects of all groups were chosen middle aged to avoid the influence of the age as a factor affecting the results.

In the present study expectedly serum FT3 was significantly higher in hypothyroid group compared to H pylori positive and control groups ($P < 0.001$ for both) and between H pylori positive and control groups ($p<0.05$). As

regard FT4 there was no significant difference between groups. The results of the mean level of TSH there was high significant difference between hypothyroid and H pylori positive groups ($P < 0.001$) and between hypothyroid and control groups ($P < 0.001$), but no statistical difference was found between H pylori positive and control groups ($p>0.05$).

Thyroid TG and TPO antibodies still retain its strong value in the screening for thyroid autoimmunity (Scherbaum, 1987).

In this study TG and TPO antibodies showed highly significant increase in hypothyroid group versus H pylori positive group ($P < 0.001$ for both) while no significant difference was found between H pylori positive and control groups ($p>0.05$).

In agreement to these results Iwazu et al., (2008) demonstrated that high titer of both TG and TPO antibodies indicated Hashimoto's disease.

Lorini et al. (2003) studied 33 patients with Hashimoto's disease that were euthyroid clinically. They found that patients with Hashimoto's thyroiditis have positive TG and/or to TPO antibodies in blood. Thyroid function could be normal or abnormal (overt hypothyroidism, subclinical hypothyroidism or hyperthyroidism).

On comparing the H. pylori antibodies titer in patients of hypothyroid with control groups, there was high significant difference between the two groups indicating the association between hypothyroidism and H. pylori infection. But there was no significant difference when comparing hypothyroid and control groups ($p>0.05$).

The study of Segni et al. (2004) showed that autoimmune gastritis is an early event in autoimmune thyroid disease with detectable parietal cell antibodies. Larizza et al. (2006) demonstrated that H. pylori might be the possible etiological factor for autoimmune thyrodites (AT) development. They found a significant interaction between HLA-DRBI*0301 and H. pylori infection in autoimmune thyrodities patients

In hypothyroid group patients with positive H.pylori antibodies (39cases) had significantly higher TG-Ab and TSH ($p<0.001$ for both) and significantly lower free T4($p<0.001$) with no significant difference in the FreeT3 level and TPO Ab($p>0.05$) compared to those with (-ve) H. pylori-Ab (10 cases),the difference in the level of TG-Ab is not due to the effect of age as there was no significant difference in the age of the (-ve) & (+ve) H. pylori-Ab subgroups. Figura et al. (1999) found a relationship between H. pylori infections& high thyroid antibodies titer, also he stated that there were controversial reports linking H. pylori infection to ATD .Similar results were found by (Tomasi et al., 2005) who suggested that monoclonal antibodies against Cag-A positive H. pylori

strains can cross react with follicular cells of the thyroid gland.

Checchi et al. (2010) found that autoimmune gastritis is frequently associated with autoimmune thyroiditis and other organ specific autoimmune diseases and may lead to atrophic body gastritis.

Sterzl et al. (2008) examined the H. pylori antibodies in patients with autoimmune thyroiditis, with and without different polyglandular involvement and compared it to healthy controls. They concluded that a different distribution of antibodies to H pylori antigens was found in patients with isolated autoimmune thyroiditis compared to patients with autoimmune thyroiditis coupled with a polyglandular syndrome.

In the present study H pylori positive group was subdivided according to presence or absence of Thyroglobulin antibodies and TPO antibodies into +ve and -ve subgroups.

According to the results of TG antibodies only 5 cases were positive and 45 cases were negative. The positive cases showed significantly higher H. pylori titer ($p<0.001$).

On the same time 7 cases had positive and 43 had negative TPO antibodies. Still with the TPO positive cases there was significantly higher H. pylori titer ($p<0.001$).

Kountouras et al. (2005) reported a strong strong relation between the levels of anti-H Pylori IgG and the level of TPO antibodies suggesting that H. pylori antigens might be involved in the development of autoimmune atrophic thyroiditis or that autoimmune function in autoimmune thyroiditis may increase the likelihood of H. pylori infection.

In H pylori positive group both the (+ve) thyroid antibodies subgroups {TG-Ab (5 cases) &TPO-Ab (7 cases)} showed significantly higher TSH with significantly lower freeT4&freeT3 compared to the (-ve) subgroups. The same results was found by Centanni et al (2006) who found increase daily doses of thyroxin in patients with H. pylori related gastritis than controls. He explained this by the fact that a normal gastric secretion is essential for effective absorption for oral thyroxin.

The previously reported association between autoimmune thyroiditis and H. pylori infection was not observed in the study of Tomasi et al. (2005) who` found that infection by H. pylori does not appear to increase the risk of autoimmune thyroiditis in individuals with dyspeptic symptoms.In agreement with our results Cammarota et al., (1997) found that there is increasing evidence for a link between H. pylori infection and the development lymphoid follicles in the gastric mucosa which are common in autoimmune thyroid disease.

It has been noted that H. pylori organism possessing the Cag pathogenicity island carried a gene encoding for an endogenous peroxidase, thus Cag. A H. pylori infection increases the risk of autoimmune thyroid disease development (Figura et al., 1999).

The results of the study of Bakos and Hillander (2003) strengthen the possibility of cross reactivity being triggered between Cag A positive H. pylori strains and autoimmune thyroiditis.

The present study showed significant positive correlation between the H.pylori antibodies titre with age, TSH, thyroglobulin antibodies titer, TPO antibodies titer and significantly negative correlation with free T4 and no correlation with freeT3.

On correlating the H. pylori Ab scoring(-ve &+ve)to all the studied parameters ,there was positive significant correlation to age ,TSH and negative correlation with free T4 but still it didn't reach the significant levels and no correlation with freeT3, thyroglobulin (+ve) antibodies and TPO (+ve) antibodies.

Bertalot et al. (2004) reported association of H. pylori infection with thyroid autoantibodies. Infection may be involved in specific autoimmune disorders such as Hashimoto thyroiditis, as suggested by Franceschi et al. (2004).

The strong correlation between IgG anti H. pylori antibodies and thyroid auto-antibodies, as well as the observation that eradication of H. pylori infection is followed by a gradual decrease in the levels of thyroid auto-antibodies, suggests that H. pylori antigens might be involved in the development of autoimmune atrophic thyroiditis or that autoimmune function in this disease may increase the likelihood of H .pylori infection. (Bertalot et al., 2004). Indeed ,the function of follicular cells of thyroid gland has been shown to be changed in H. pylori infection, eradicated H .pylori infection in autoimmune thyroiditis has been ascribed at least in part to all or some of decreased the morbidity of autoimmune thyroiditis (Bertalot et al (2004).

It is well known that male gender was a factor associated with prevalence for H. pylori infection (Replogi et al., 1995). The possible difference of H. pylori prevalence is unclear but may relate to young boys having poorer hygiene than young girls (Kikuchi and Dore, 2005).In this study, in group II 13(27%) of patients with positive H.pylori Ab were males while 37(74%) were females. On the other hand, Goh (1997) noted that the importance or even the existence of differences in rates of infection with H. pylori between sexes is uncertain. It is an exceedingly common infection with an estimated overall

prevalence of infection in middle-age adults of 74% in developing countries and 58% in developed countries .The prevalence of infection increases rapidly with age (Kelsen et al., 2008).

There are a number of possible reasons for the differences found between the results presented in the present study and those found elsewhere. First of all is that there is a geographic variation in the risk of thyroid dysfunction between regions as subjects of this study were Egyptian , which could reflect a variety of environmental and/or genetic factors (Barker and Phillips, 1984). The difference may reflect the methodologies employed. Some have used population screening and follow-up of survivors' incident cases from abnormal biochemistry results or hospital admissions (Berglund et al., 1996). The population dynamics of the different locations also have an impact on the levels of thyroid dysfunction, because incidence was higher among females and the elderly.

Conclusion

This study revealed that patients with positive TTG & TPO antibodies , showed (+ve) H.pylori AB, with significant high titre in their sera. The patients with positive H. Pylori AB showed high serum titre of TG-AB. In our study H.pylori-AB correlates to thyroid function tests and thyroid antibodies.

Recommendations

It is recommended that patients with helicobacter pylori infection should be followed up regularly by repeated tests of TG-AB and TPO-AB autoantibodies in their sera, for earlier detection of subclinical autoimmune thyroid dysfunction before any changes in TSH values might occur.

It is recommended also to consider that patients with autoimmune thyroid dysfunction should be followed up for detection of H.pylori infection even without dyspeptic symptoms because there is positive correlation between H.pylori infection and autoimmune thyroid disease especially those on high doses of thyroxin.

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الارتباط بين عدوى البكتيريا الحلزونية البوابية و مرض نقص هرمون الغدة الدرقية المناعى

فاطمه كامل حماد , زينب عبد الباسط حسن , دينا محمد أباظة ,سبيلة جمعة موسى , أحمد أحمد سعد و هشام محمد أبو السعود
قسم الغدد الصماء و الأيض ,قسم الباطنة العامة[2] كلية طب بنات الازهر. ,قسم الباطنة العامة كلية طب جامعة القاهرة

التهاب الغدة الدرقية المناعى يعتبر من أكثر المسببات لأمراض الغددة الدرقية بالنسبه للبالغين خاصة النساء. وقد وجد أن هناك العديد من اختبارات الأجسام المضادة للغده الدرقيه و الأجسام المضادة للثيروجلوبيولين و التى تعتبر مؤشرا هاما لتشخيص أمراض الغدة الدرقية المناعية.
و وجد أن ميكروب البكتيريا الحلزونية البوابية يلعب دورا هاما فى الاصابة بالتهاب المعدة المزمن،و أمراض القرحة و سرطان المعدة
إن الميكروب الحلزونى و المعروف بدوره فى مرض قرحة المعدة و قرحة الإثنى عشر له أيضا دورا فى حدوث تأثير على الأعضاء الأخرى للجسم بعيدا عن الجهاز الهضمى.
لقد وجد أن المرضى المصابون بالتهاب الغدة الدراقية المناعى يكونون معرضين بصورة كبيرة للإصابة بميكروب البكتريا الحلزونية البوابية مما يعطى احتمال أن الأجسام المضادة لهذا الميكروب من الممكن أن تتفاعل مع الخلايا الحويصليه للغده الدرقية.

الغرض من البحث

دراسة الارتباط بين عدوى البكتريا الحلزونيه البوابيه و نقص هرمون الغده الدرقيه المناعى.
إشتملت الدراسه على 147 حاله تم اختيارهم من العيادات الخارجيه تتراوح أعمارهم بين 20-50 عاما.
تم تقسيم المرضى الى 3 مجموعات كالأتى:

- المجموعه الأولى: اشتملت على 49 مريض (13 من الذكور و 36 من الإناث) يعانون من نقص فى وظائف الغده الدرقيه بدون أى أعراض تشير إلى الإصابه بالميكروب الحلزونى.
- المجموعة الثانية: اشتملت على 50 مريض (13 من الذكور و 37 من الإناث) يعانون من أعراض الميكروب الحلزونى و لا يوجد لديهم أى شكوى من أمراض الغده الدرقيه.
- المجموعه الثالثه: اشتملت على 48 شخص (17 من الذكور و 31 من الإناث) كمجموعه ضابطه.

لكل المجموعات تم عمل الآتى:

- اخد تاريخ مرضى بالتفصيل مع كشف طبى كامل.
- الكشف عن مستوى هرمونات الغده الدرقيه فى الدم TSH,FT4,FT3
- الكشف عن الاجسام المضادة و هي H. pylori-Ab, TPO-Ab,TG-Ab

نتائج البحث

- أن المرضى المصابين بارتفاع فى نسبه الأجسام المضادة للغده الدرقيه لديهم ارتفاع فى نسبه الأجسام المضاده للميكروب الحلزونى.
- إن المرضى المصابين بارتفاع نسبه الأجسام المضادة للميكروب الحلزونى لديهم ارتفاع فى نسبه الأجسام المضادة للغدة الدرقيه.

وجد أن هناك علاقه طرديه بين ارتفاع نسبه الأجسام المضادة للميكروب الحلزونى و الأجسام المضادة للغدة الدرقية.
أن النتائج هذا البحث تتفق مع اراء الباحثين من قبل فى هدا الموضوع حيث بين بعضهم أن الميكروب الحلزونى له دور فى أمراض الغده الدرقيه و خاصه الأمراض المناعيه للغدة الدرقيه.