

International Journal of Surgery Science

E-ISSN: 2616-3470

P-ISSN: 2616-3462

© Surgery Science

www.surgeryscience.com

2020; 4(1): 470-472

Received: 20-11-2019

Accepted: 22-12-2019

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Clinical profile of patients with Hashimotos thyroiditis

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DOI: <https://doi.org/10.33545/surgery.2020.v4.i1h.450>

Abstract

Autoimmune responses against specific antigens are primary determinants in thyroid autoimmunity. Other molecular mechanisms including cell apoptosis may play a role in determining the opposite phenotypic outcomes of AITD such as thyroid destruction in HT and thyroid hyperplasia in GD. T-helper lymphocytes produce cytokines that influence both immune and target cells at several levels. 100 consecutive patients with diagnosis of Hashimoto's thyroiditis were included in this study. Detailed clinical history and physical examination of the patients was done. Suspected patients were subjected to thyroid function test, FNAC, USG neck. The diagnosis was confirmed with serology. Age of the patients varied between 12yrs. The mean age of the patients in this study was 39 yrs. Of all the patients with Hashimotos thyroiditis, 6 patients were male and 94 patients were female. The maximum number of patients presented with lethargy, weakness and other non-specific features of hypothyroidism a significant cohort resented with menorrhagia as their chief complaint about 9% patients had goiter and 06 of them also had pressure symptoms.

Keywords: Clinical profile, Hashimotos thyroiditis, Goiter

Introduction

Autoantibodies against other thyroid-specific antigens such as thyrotropin receptor and sodium iodide symporter were also found in serum of HT patients. However, these antibodies occur at low frequency and do not appear to contribute any diagnostic power.

In a final, destructive step of Hashimoto's thyroiditis, the auto reactive T cells diffusely accumulate in large numbers and infiltrate thyroid parenchyma (Fig. 2). In the BB-DP rat model, T-helper type 1 (TH1)-mediated mechanisms involving production of IL-12, tumor necrosis factor- α (TNF- α) and interferon- γ play a major role in the destruction of thyrocyte, rather than TH2 type mechanisms. Fas and Fas ligand (FasL) expression was higher in rats with lymphocytic thyroiditis indicating a role of these apoptotic molecules in thyrocyte death ^[1].

Autoimmune responses against specific antigens are primary determinants in thyroid autoimmunity. Other molecular mechanisms including cell apoptosis may play a role in determining the opposite phenotypic outcomes of AITD such as thyroid destruction in HT and thyroid hyperplasia in GD. T-helper lymphocytes produce cytokines that influence both immune and target cells at several levels. The predominance of TH1 or TH2 cytokines might regulate thyrocyte survival through the induction of pro-apoptotic and anti-apoptotic proteins. TH1-mediated mechanisms lead to thyrocyte ^[2].

Depletion in Hashimotos's through the involvement of death receptors and cytokine-regulated apoptotic pathways. The normal thyroid gland has been shown to act as an immune privileged site having carefully regulated mechanisms of cell death and self-protection against attack by infiltrating activated T-cells induces by apoptosis. Cell apoptosis occurs in the normal thyroid at a low level. As new thyrocytes are produced, old cells are destroyed in order to maintain normal thyroid volume and function. Deregulation of apoptosis, which is weakly determined by genetic susceptibility, can lead to destructive processes. Initiation of an out-of control apoptotic mechanism in thyroid cells may be caused by various non-genetic injuries that affect expression of apoptosis inhibitor molecule Bcl-2 or membrane ligand FasL. Thyrocytes from HT thyroid glands are able to *heeb produce* Fas and FasL on their surfaces thus inducing fratricide apoptosis. IL-16, abundantly produced in HT glands, induces Fas expression in normal thyrocytes, the cross-linking of Fas resulting in massive thyrocyte apoptosis. This can play a role in the progression of Hashimoto's thyroiditis ^[3, 4].

Immune-mediated apoptosis of thyrocytes is directed by CD8+ cells. Receptors on the target cell are triggered by lymphocyte ligands and /or released soluble factors are delivered to the target

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cell. Receptors involved in immune-mediated apoptosis include the TNF R1 receptor, the Fas receptor and death receptors DR3 and DR4, whereas soluble mediators include substances such as perforin and TNF.

The common apoptotic pathway consists of subsequent activation of specific intracellular proteases known as caspases. These caspases are themselves activated by specific proteolytic cleavage or may be activated by cleavage performed by other caspases. The caspase cascade ultimately induces enzymes that progressively destroy the cell and its genetic material, finally lead to cell death. The apoptosis, or programmed cell death, can be initiated by binding death, can be initiated by binding death ligands, such as TNF, TNF-related apoptosis-induced ligand (TRAIL) and FasL, to the cell surface. This in turn starts intracellular signal cascading of caspases [5].

Several apoptosis signaling pathways, initiated by molecules such as Fas L and TRAIL, have been shown to be active in thyrocytes and may be involved in destructive thyroiditis. Fas-mediated apoptosis seems to be a general mechanism of cell destruction in AITD. In GD patients, reduced levels of Fas/Fas L and increased levels of Antapoptotic molecule Bcl-2 favor thyroid cell survival and apoptosis of infiltrating lymphocytes. In contrast, the regulation of Fas/FasL Bcl-2 expression in HT can promote thyrocyte apoptosis through homophylic Fas-FasL interactions and a gradual reduction in thyrocyte numbers leading to hypothyroidism [6].

Thus, the rate of thyrocyte apoptosis dictates the clinical outcome of thyroid autoimmunity. Though rare in normal thyroid, it markedly increases during HT, but not in GD.

Therefore, regulation of thyrocyte survival is a crucial pathogenic determinant.

Methodology

The present study was carried out on patients who visited hospital and were diagnosed to have Hashimoto's thyroiditis.

100 consecutive patients with diagnosis of Hashimoto's thyroiditis were included in this study. Detailed clinical history and physical examination of the patients was done. Suspected patients were subjected to thyroid function test, FNAC, USG neck. The diagnosis was confirmed with serology.

After confirmation of diagnosis, patients were treated with levothyroxine at the standard dose of 1.6-1.8 mcg/kg lean body weight per day. A subset of patients was treated surgically.

The criterion for surgical intervention was

- Presence of a nodule suspicious of malignancy
- Large goiter causing compressive symptoms
- Painful Hashimotos
- Cosmetic

All candidates accepted for operative treatment underwent total thyroidectomy as per internationally accepted guidelines. The specimens of operated cases were routinely subjected to HPE examination to detect the associated pathologies, particularly malignancies. All patients in post-operative phase were put on levothyroxine.

Results

Table 1: Age distribution

Age in years	No. of patients
10-20	1
20-30	20
30-40	42
40-50	25
50-60	9
>60	3
total	100

Age of the patients varied between 12yrs. The mean age of the patients in this study was 39 yrs.

Of all the patients with Hashimotos thyroiditis, 6 patients were male and 94 patients were female.

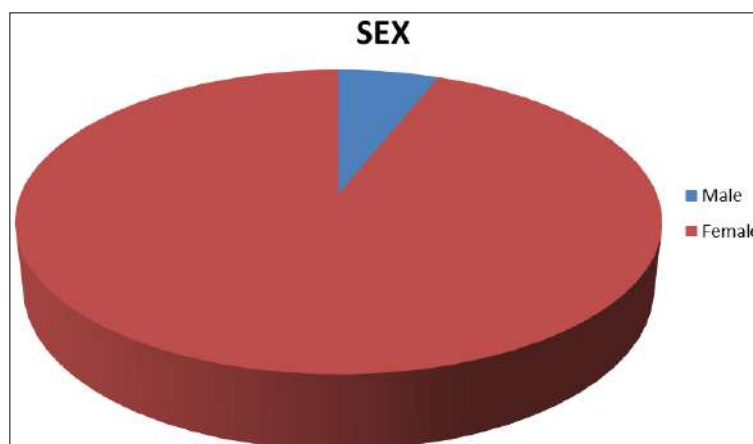


Fig 1: Sex distribution

Table 2: Presenting Symptoms

Presenting symptoms	Number of patients	Percentage
Lethargy/ weakness/mental slowing	90	90
Menorrhagia	20	20
Pain	15	15
Goitre	09	9
Pressure symptoms	06	6
Orbitopathy	01	1

The maximum number of patients presented with lethargy, weakness and other non-specific features of hypothyroidism a significant cohort resented with menorrhagia as their chief complaint about 9% Patients had goiter and 06 of them also had pressure symptoms. This can be attributed to the fact ours being tertiary hospital, complicated goitrous presentation is likely to predominate. One patient presented with orbitopathy which is indeed a very rare presentation of Hashimotos thyroiditis.

Discussion

Table 3: Age incidence seen in our study and other studies

Study	Our study	Siriweera <i>et al.</i> [7]	Erdogan <i>et al.</i> [8]	Jayaram <i>et al.</i> [9]
Peak age	39 yrs	4.3 yrs	41.76 yrs	38 yrs

In the present study, the peak incidence was seen at the age of 39 yrs. Most of the larger international studies reported above show similar findings indicating that HT is most commonly seen in middle aged women.

Table 4: Sex incidence in other studies and present study

Study	Present study	Siriweera <i>et al.</i> [7]	Erdogan <i>et al.</i> [8]
Female%	94%	91.12%	96%

In the present study 94 percent patients were females.

The major presentation of our patients was with non-specific symptoms of lethargy and tiredness indicating altered thyroid status

Table 5: Presenting Symptoms

Presenting symptoms	Number of patients (present study)	Ray <i>et al.</i> , 2009 [10]
Lethargy/ weakness/mental slowing	90	75
Menorrhagia	20	25
Pain	15	09
Goitre	09	27
Pressure symptoms	06	09
Orbitopathy	01	01

As compared to the other studies, prevalence of goiter in our study appears to be less. This can be explained by the fact that most of these studies have been carried

Out in iodine deplete areas of the world where the incidence of goitrous presentation is high.

Conclusion

This disorder occurs more commonly in females. It is more common in middle aged females. Most women present with signs of hypothyroidism.

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