Introduction

There are two ways to diagnose a disease involving organs and/or tissues: by morphological detection or by functional evaluation. Several modern imaging modalities play a role in the morphological detection of disease, including ultrasonography, computed tomography, magnetic resonance imaging, computed radionuclide imaging and angiography. For the functional assessment of diseases, a number of *in vitro* procedures can be carried out in a well set-up laboratory, such as analysis of serum free thyroxine level (FT4) in the evaluation of thyroid function. Furthermore, in certain circumstances an imaging modality can also be used to evaluate the function of an organ, such as left ventricular stroke volume and cardiac output using contrast ventriculography.

High-resolution real-time ultrasonography is currently accepted world-wide as a reliable method in the morphological evaluation of thyroid diseases [1–6]. However, for the diagnosis of diffuse toxic goiter (i.e. Graves’ disease), both serum FT4 and thyroid-stimulating hormone (TSH) levels should be measured [7]. For the diagnosis of Hashimoto’s thyroiditis, in addition to FT4 and TSH, the antibody titers to thyroglobulin and thyroid peroxidase are also required [7]. Since Graves’ disease usually leads to a hyperthyroid state, and the majority of
Hashimoto’s thyroiditis patients are usually in a clinically hypothyroid state, the hemodynamic changes in the cardiovascular system can be an important sign in differentiating these two diseases [7]. Using color Doppler ultrasonography (CDUS), intra-glandular hypervascularity was reported in diffuse thyroid diseases, irrespective of the hyperthyroid, euthyroid or hypothyroid state/phase [8–21]. Alteration of the hemodynamic kinesis in the systemic circulation cannot explain the latter condition. However, why there is increased parenchymal vascularity in various diffuse thyroid diseases in spite of the different functional states or phases is unknown.

Why Hypervascularity in Diffuse Thyroid Diseases?

Graves’ disease and Hashimoto’s thyroiditis are autoimmune disorders [7]. Could the hypervascular pattern, shown as increased color flow by CDUS in previous reports [8–21], be an inflammatory reaction to autoimmunity of the gland in these diseases? Several angiogenic factors, including vascular endothelial growth factor [22] and chemokine CXC ligand 10 [21], may play an important role in activating intrathyroid angiogenesis. TSH elevation and increased TSH receptor antibodies might be the trigger in the mechanism of angiogenesis in Hashimoto’s thyroiditis [9,11,23–25]; however, in Graves’ disease serum TSH is usually low [7]. Both serum anti-thyroglobulin and anti-thyroid peroxidase antibodies may be increased in Graves’ disease and Hashimoto’s thyroiditis [7], although they do not seem to be the main factors in angiogenesis [25]. Therefore, the true mechanism of hypervascularity in diffuse thyroid diseases is still unknown.

Spectral Doppler Analysis in Diffuse Thyroid Diseases

Measurement of the peak systolic velocity (PSV) in the superior and/or inferior thyroid arteries with spectral Doppler analysis (SDA) also demonstrates the hemodynamic changes in the thyroid gland [12–15,21,25–29]. There is a positive correlation between the qualitative parenchymal blood flow assessment by CDUS and the quantitative measurement of PSV by SDA of the thyroid gland [21,25,28]. Increased PSV of thyroid arteries was evidenced in Graves’ disease [9,12–16,19,20,22,23,25,27–29] with hyperthyroidism resulting in tachycardia and high cardiac output [7]. However, PSV elevation in the thyroid arteries was also evidenced in Hashimoto’s thyroiditis [11,18,22–25,27] and other thyroid diseases, such as simple goiter and goiter with non-functioning nodule(s) [27], with the possible causative etiologies of increased thyroid volume [24,27] and autoimmune inflammatory processes [9–11, 22–25], rather than increased cardiac output itself. Thus, increased thyroid arterial PSV alone is not a reliable sign of hyperthyroidism [25], and is inadequate for the assessment of thyroid function.

In addition to thyroid arterial PSV measured by SDA, resistive index has also been reported to be valuable as a functional index in the follow-up of medical treatment of Graves’ disease [30]. Yet, it was recently considered inappropriate in the evaluation of post-treatment follow-up of either hyperthyroidism [29] or hypothyroidism [25].

Peak Systolic Velocity of Common Carotid Artery: A Complementary Indicator of Thyroid Function

In cases without cardiovascular anomaly, the thyroid functional states may influence cardiovascular hemodynamics, resulting in changes in stroke volume, heart rate and cardiac output [7]. Hyperthyroidism is usually associated with increased cardiac output, tachycardia and, thereafter, increased systemic arterial blood flow [7]. In contrast, hypothyroidism manifests with decreased stroke volume, cardiac output, and bradycardia [7]. Increased cardiac output usually leads to rapid pulse rate and elevated arterial flow velocity, while decreased cardiac output leads to reduced pulse rate and arterial flow.
velocity. Therefore, measuring flow velocity in the aorta and its main arterial branches in patients without significant cardiovascular anomaly could be of value in the evaluation of thyroid function.

The common carotid arteries are the major branches of the thoracic aorta; their hemodynamic changes directly reflect the aortic flow kinetics and cardiac output. Observations of their hemodynamics by CDUS and SDA are easy and convenient prior to ultrasonographic examination of the thyroid, because they are close to the gland. A previous ultrasonographic study with CDUS and SDA revealed that increased PSV of the common carotid arteries occurred in more than 70% of cases with Graves’ disease, and less than 10% of cases with goiter with nodules and thyroiditis [27]. The results suggest that increased PSV of the main arteries in the systemic circulation, in combination with increased thyroid arterial PSV, can be a good indicator in differentiating Graves’ disease from other nodular and diffuse thyroid disease including Hashimoto’s thyroiditis. Accordingly, it could be of value to perform a further ultrasonographic study involving a large number of cases to assess the functional states of the thyroid, by measuring the PSV in both the thyroid arteries and the common carotid arteries, in the differential diagnosis of these two diffuse autoimmune thyroid diseases.

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References


