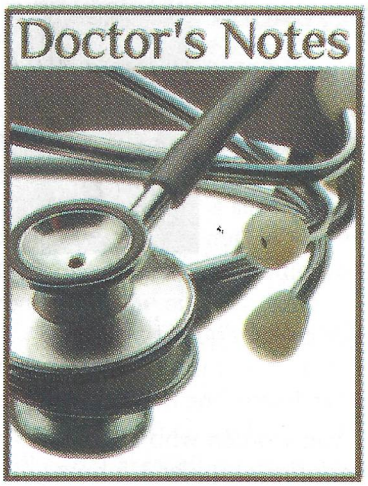

Environmental Processes in Thyroid Disease

Over the last sixty years, most forms of auto-immune disease, including thyroid disease, have been on the rise throughout many industrialized nations to such an unprecedented extent that they have become the third leading category of illness causing sickness and death in the United States. But this modern scourge is not without a face. Many articles have helped to illustrate that among the multi-factorial nature of auto-immune disease, environmental toxins stand out as a well-documented contributor to havoc in the immune system.

The thyroid gland is an endocrine organ which serves as the thermostat for the body, overseeing many metabolic functions and processes. Auto-immune thyroid disease comes in two forms: Graves' disease and Hashimoto's thyroiditis. Graves' disease, or hyperthyroiditis, occurs when antibodies misguidedly attack and over-stimulate the thyroid gland, resulting in a corresponding excess of metabolic and excitatory stimuli such as palpitations, sweating, and weight loss. Hashimoto's thyroiditis is the opposite extreme whereby antibodies attack the thyroid, resulting in decreased function and 'hypothyroidism' with symptoms such as intolerance to cold, depression, and weight gain. There are over 5 million Americans with auto-immune thyroid disease, two thirds of whom have Graves' disease and one third with Hashimoto's. Though epidemiologic data is comparatively limited, it does seem that incidence and prevalence of both these conditions has been increasing, irrespective of advances in diagnosis and detection. By far the majority of these cases continue to be among women, as much as a 3:1 incidence when compared with men.

Auto-immune thyroid disease, like most other auto-immune diseases, does not develop suddenly but comes on gradually, through an extended germination period, before becoming clinically observable. This is evidenced by the millions of people that test positive for ANA (anti-nuclear antibodies) each year before they have any overt symptoms. Furthermore, studies have shown individuals with a higher number of auto-antibodies present have a higher risk of developing a disease within five years. Thus it would seem there are additional environmental factors that dampen or amplify the pathological process over time.

We must then look to the process itself for answers. Evidence has shown that triggers for auto-immune reactions exist as cell stressors which threaten the integrity of the tissue in question. The list of cell stressors includes, but is not limited to, such physical stressors as heat or radiation, biologic stressors such as infection or inflammation, osmotic stressors such as changes in concentrations of salts and sugars, antibiotics, alcohols, metals, and a host of chemical compounds. If these triggers find themselves in an environment with the proper antecedent conditions of genetic susceptibility and lifestyle, then the odds for disease increase. Furthermore, if the person does not have sufficient mediators to turn this process around - antioxidants and omega-three fatty acids to name a few - then they slip further down the slope of chronic disease.

The upshot of this for auto-immune thyroid disease is that there are several points where one has the ability to attempt to turn the tide away from imbalance and further deterioration. The wide array of cell stressors and triggers contains a broad category of environmental toxins, which is particularly noteworthy because here we have the opportunity to exert more control over our destiny than in many other areas. Additionally, science has the ammunition to back this up. Thyroid disease has been associated with exposure to polybrominated biphenyls (PBBs), polychlorinated biphenyls (PCBs), thiocyanates (in cigarette smoke), chlorine and fluoride (in water), as well as medications such as lithium, penicillamine, and amiodarone.

However, the environmental contaminants receiving the most attention in recent years are the endocrine disruptor 'xenoestrogens,' which are hormonally active agents in the environment. The full extent of the impact of these has yet to be determined, yet their concentrations measured in random blood samples have shown them to be pervasive and persistent in the body. These compounds are of particular interest because of their ability to mimic estrogen, which is higher in females than males (as is auto-immune disease in general) and particularly higher when women are in their 20's, which is when there is an increased incidence of auto-immune disease diagnosis. Common xenoestrogens include organochlorine pesticides (dieldren, DDT, aldicarb, pyrethroids) dioxins, polychlorinated dibenzofurans, bisphenol-A, phthalates and other plasticizers, parabens, steroids given to livestock, mycotoxins, and DES.

In the face of ever-increasing incidence of thyroid and auto-immune disease, there is still information which we can use to give ourselves the best possible outcomes. If you or someone you know was recently found to have a positive ANA or other auto-antibodies without a clear diagnosis, rather than adhering to the conventional advice of 'watching and waiting,' implore them to get busy.

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