Autoimmunity: The normal immunological control mechanisms when outlined above become defective.

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Background

Autoimmune reactions and illnesses suggest that the typical immunological regulatory systems discussed above go faulty in specific conditions. This aberrant reaction to "self-antigens" is thought to be caused by a number of ways. Cross reactivity, molecular mimicry, T cell epitope provision, release of sequestered or cryptic antigens, failure of T cell control and anti-idiotype reactions are some of these. The traditional link between group A streptococcal infection and rheumatic heart disease is due to "cross reactivity," in which streptococci release antigens that are structurally similar to self-antigens on cardiac muscle. Antibodies and T cells raised specifically against the streptococcal antigen destroy structurally identical self-proteins as a result [1].

The concept of "molecular mimicry" is also proposed at the molecular level. Short molecular sequences (for example, 5-amino-acid sequences) are known to be shared by microbes and self-proteins. Both of these mechanisms are thought to play a role in the development of auto reactivity. Another technique for bypassing normal immune control is the potential attachment of foreign proteins (e.g. medicines or chemicals) to self-proteins. B cells that bind to the self-non-self-complex have the ability to digest the non-self-component and present it to T cells that are reactive to foreign proteins. As a result, T cells may send out improper helper signals to B cells that attach to the self-component, triggering an autoimmune response. The process is referred to as "provision of T cell epitopes." Furthermore, some self-antigens, such as lens proteins from the eye, are not ordinarily accessible to immune system cells and are considered to be "sequestered." Tissue damage can release antigens that trigger an immune response, for example, after traumatic damage to one eye, proteins released can cause autoimmune damage to the other - sympathetic ophthalmia [2].

Antigens that are only released during the natural turnover of body proteins by antigen presenting cells are known as cryptic antigens. Tolerance does not develop because they are not routinely produced, and their release can lead to autoimmune responses. It's likely that cryptic epitopes are only released in low amounts in the wild; nonetheless, coincidental variables like infection or inflammation may be required to trigger an autoimmune response. Failure of T cell control (or suppression) has long been thought to be a mechanism for the development of auto reactive responses. Because the form of an immune response to a microbe is influenced by the balance of cytokines produced in the microenvironment, it is considered that the balance of T cell cytokine production in humans may be crucial in affecting autoimmune reactions. Finally, idiotypes are the antigen binding sites of antibody molecules, and it's probable that during a typical infection response, a "second wave" of anti-idiotype antibodies is produced, directed not against the microbe but against these idiotypic sites, resulting in auto antibodies [3].

The importance of central tolerance and effective regulatory T cell mechanisms is shown by two diseases [4]. Mutations in the autoimmune regulator gene (AIRE) on chromosome 21 cause Autoimmune PolyEndocrinopathy-Candidiasis-Ectodermal Dysplasia syndrome type 1 (APECED Type 1). These mutations cause autoreactive T cells to fail to apoptose, resulting in the development of autoimmunity. Mutations in the forkhead box P3 (FOXP3) gene in regulatory T cells are seen in patients with IPEX (Immunological dysfunction, Polyendocrinopathy, Enteropathy, X-linked) disease, which leads to severe autoimmunity and immune insufficiency.

In clinical terms, it's vital to distinguish between autoimmune reactions (which might happen as a result of infection, surgery, drug therapy, or getting older and are marked by the presence of autoantibodies in the blood) and autoimmune illness.

The immune system may be able to restore control of autoreactive lymphocyte clones after the "external shock" has been removed, indicating that auto reactivity is just transient. Autoimmune reactions, rather than autoimmune disease, are far more common. The presence of autoantibodies in the blood, even in an ill patient, does not always imply that the patient has an autoimmune illness; for example, 10% of people over the age of 70 have detectable antinuclear antibodies in their serum. Only when tissue damage and symptoms arise, as well as autoreactive antibodies or T cells, is autoimmune disease diagnosed [5].

Autoantibodies are classified as either primary or secondary. Primary autoantibodies, which are characterised as producing disease directly, are uncommon in clinical practise. Antibodies against the TSH receptor in Graves' disease, anti-acetylcholine receptor in Myasthenia gravis, and anti-voltage gated calcium channel blocking antibodies in Lambert Eaton Myasthenic syndrome are just a few examples. Secondary autoantibodies

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develop as a result of an autoimmune response, may be linked to a specific disease (and so be diagnostically useful), but do not cause disease directly. Antinuclear antibodies in systemic lupus erythematosus and antimitochondrial antibodies in primary biliary cirrhosis are examples of secondary autoantibodies.

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