T-Cell Ratios: Modulation by Nutrition: Case Report

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Interest in T-cell function has been stimulated by the immunological abnormalities in AIDS patients. These patients are marked by abnormalities in T-cell or thymus cell function, such as cutaneous anergy, lymphopenia, and a decrease in T-cell proliferative response to mitogens, but most strikingly, a decreased number in the percentage of T-helper cells (Cohen et al, 1986).

T-cells are particularly valuable in relationship to the control of viral and fungal infections. T-cells have a direct role in direct and indirect control of bacterial infections (Blumberg and Schooley, 1985; Braverman and Pfeiffer, 1982). Severe defects in T-lymphocyte functions can lead to an increased susceptibility to viral, fungal and bacterial infections and may also be an indicator of the degree of exposure to environmental carcinogens and toxins (Blumberg and Schooley, 1985). Most T-lymphocyte-immuno deficiencies are due to intrinsic abnormalities in the lymphoid-stem cells (Blumberg and Schooley, 1985). The only curative therapy for T-lymphocyte defects and genetic diseases is the replacement of the normal lymphocyte stem cells. Bone-marrow transplantation is the primary therapy for T-lymphocyte defects. Bone-marrow therapy may have a future role in AIDS and other patients with T-helper-cell deficiencies.

I began studying T-cell ratios (T-helper/ T-suppressor) because of the increasing number of patients that I saw complaining of viral-like illness of unknown etiology, i.e., sore throats, low-grade fever, weakness, persistent fatigue, and swollen glands. About 50% of these patients have had T-cell abnormalities. Decreases in T-helper cells are found in viral illness and chronic disease. There may be a new syndrome a pre-AIDS related complex (pre-ARC). Increases in T-helper cells occur in autoimmune diseases, healing ulcers, and forms of Leprosy (Table 1). T-helper cell deficiency is likely to be an increasing problem even in non-AIDS patients.

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Case History: Intermittent Viral Illness

A thirteen year-old, blond, blue-eyed, 5'9" male, 120 lbs., BP 80/50, P 60, presented to us, complaining of intermittent viral-like infections, unresponsive to antibiotics and marked by a low-grade temperature, upper respiratory symptoms, stomach pains and drowsiness. He had a loss of appetite, craving for sweets, and allergies to dust. The child had missed 50% of the school year and had almost been left back.

His initial treatment (3/85) was 20 mg of zinc, elemental as the sulfate, 50 mg of vitamin B-6, 4000 mg of vitamin C, 400 micrograms of selenium, and a multi-vitamin Willvite daily. When he returned for his next visit one month later, he showed no improvement, weighing 121 pounds, with blood pressure 80/55. The following tests results were noted: plasma histamine of 52.6 ng/ml, copper 95 meg % (normal 80-120 mcg%), zinc 78 mcg % (normal 100-120 mcg%), iron 58 mcg % (normal 40-120 mcg%), urinary kryptopyrrole (KP) 21 mcg%. He showed an IgG of 657 mg/dl, which was low (normal 750-2000), and IgA 42 mg/dl which was low (normal 80-441 mg/dl). IgM was normal. Zinc dosage was increased to 40 mg a day and vitamin B-6 was increased to 200 mg a day (4/85). He was started on deanol, 200 mg per day. Eicosopentoic Acid (EPA) was begun at 1000 mg per day but was not tolerated (nausea). He returned four months later with overall improvement. He finished summer school successfully and had gone two months without an illness or relapse (9/85). A T-cell study was done (9/85) which showed a helper-suppressor ratio of 0.9. T-helper cells and B-cells were depleted. Zinc was increased to 100 mg and later increased to 120 mg per day.

The patient reported notable improvement (11/15/85) and was now six feet tall at 137 pounds with blood pressure 90/54. He

Table 1
Ratios of T-Helper to T-Suppressor Ratios in Various Diseases

Disease	T-Cell Ratios	T-Cell Defect*[3])
Control	1.73 ±0.83	
Rikers Island Inmates with leukopenic anergy	0.31	T-helper decrease
Aphthous Stomatitis - ulcer[34] (tissue site)	0.1	T-helper decrease
Sinus Histocytosis X	0.9	T-helper decrease
2-4 Yrs. Post-Thymectomy (MG)[12]	2.1 ± 0.4	T-helper decrease?
2-6 Yrs. Post-Thymectomy (MG)[12]	2.1 ± 0.3	T-helper decrease?
6 Yrs. Post-Thymectomy (MG)[12]	2.3 ± 0.4	T-helper decrease?
Post-Thymectomy-MG[5,6]	2.0	T-helper decrease
Virus Infection - CMV, Echo-virus, Hepatitis, Mumps[7]	0.5 - 1.0	T-helper decrease
Renal Transplant-Epstein-Barr Virus, Herpes Simplex Virus[7,14]	1.0	T-helper decrease
AIDS	0.4 - 0.8	T-helper decrease
Drug Abusers[9]	0.73 ± 0.36	T-helper decrease
Dioxin Exposure (severe 10%)[21]	1.0	T-helper decrease
Pelvic Irradiation[32]	0.5-1.0	T-suppressor increase
Chronic Lymphocytic Leukemia[31]	0.5-1.0	T-suppressor increase
Rheumatoid Arthritis[16]	2.36 ± 0.67	T-helper increase
Systemic Lupus	2.41 ± 0.91	T-helper increase
Myasthenia Gravis (MG)[5,6]	2.2 + 0.2	T-helper increase
Toxic Epidermal Necrolysis[30]	2.0	T-helper increase
Aphthous Stomatitis-healing ulcer (tissue site)[34]	10:0	T-helper increase
Erythemia Nodosum Leprosum (ENL)(skin)[L27]	2.1 ±0.4	
Non-ENL Leprosy (skin)[1]	0.6 ± 0.4	T-suppressor decrease
Tuberculosis[7,36]	2.0	T-suppressor decrease
Hypergammaglobulinemia[7-25]	2.9	T-suppressor decrease
Lupoid Hepatitis[7]	2.0	T-suppressor decrease
Parasites	1.0 ± 1.5	T-suppressor decrease

(a) The primary T-cell defect is listed although secondary defects are common, i.e. T-helper decrease can be accomplished by either relative excess or deficiency of T-suppressor cells. 1 Bach et al, 1981.
5 Berrihetal, 1983.
6 Berrihetal, 1981.
7 Blumberg and Schooley, 1985.
9 Carboneetal, 1985.
12 Cox et al, 1986.
16 Fossaluzza et al, 1985.
27 Merotetal, 1986.
30 Modlinetal, 1985.
31 Perri and Key, 1986.
32 Petrinietal, 1985.
34 Savage et al, 1985.

missed only five days in the first 2 1/2 months of his school year. He had a T-cell ratio of 1:2, while his IgA was still low but increased to 60. He continued to do well and on his last visit (4/1/86) his T-suppressor ratio was 1.4 with a T-helper cell ratio of 48% (normal 32-50), and B-cells normal at 10%.

Case 2

A 24 year old male on Acutane (.5mg/kg day for 2 months) developed a flu-like syndrome which occurred intermittently for several weeks. On recovery, we checked his T-cells. T-helper was 55% (32-50% normal) with a T-helper to T-suppressor ratio of 2.4. Acutane has been helpful in a variety of illnesses including immunosuppressive diseases (Fontana et al, 1986; Goldman, 1984; Katz, 1986; Meyskens et al, 1985; Meyskens, 1983). Acutane (a vitamin-A derivative) may stimulate T-helper cells. In vitro, retinol suppresses T-lymphocyte functions. Yet, increased cancer risk, immunosuppressor effects and low vitamin A levels in serum have been noted by Watson et al, (1985).

Discussion

Approaches to T-helper deficiency have been injections with gamma globulin (Gupta et al, 1986) and thymus extract (De Martino et al, 1985) (2 mg for 9 weeks did not help a similar patient). The benefit of acutane may be due to some interaction with zinc metabolism, which is intimately linked, i.e. retinol-binding protein is zinc-dependent; hence, zinc deficiency can cause a relative vitamin-A deficiency (Goodman, 1984; Cousins and Swerdel, 1985).

Fraker and colleagues in 1978 reported that a dietary deficiency of zinc can cause rapid atrophy of the thymus and impaired T-helper-cell function. Their data and others (Baer et al, 1985) showed that zinc-deficient young animals can have T-helper cell function restored upon nutritional repletion. Braverman and Pfeiffer (1982) reviewed the critical role of zinc in the immune system, particularly in thymus cell function. There is evidence that the offspring of zinc-deficient mice are also immunodeficient (Beach et al, 1982). It has been suggested that transient hypogammoglobulinemia in infancy is a manifestation of maternal zinc deficiency (Lentz and Gershwin, 1984). This patient's initial low serum zinc and decreased IgA may be the cause of his T-helper-cell abnormality (Lentz and Gershwin, 1984). Zinc deficiency in AIDS is not uncommon (Weiner, 1984).

Clinicians that do find T-helper-cell deficiencies in patients should consider megazinc therapy, in combination with Beta-Carotene.

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