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CHESSER-NAUGLE International Lectureship Grant:

The Fungal/Mycotoxin Connections: Autoimmune Diseases, Malignancies, Atherosclerosis, Hyperllpldemias, and Gou

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TWENTY-EIGHTH ANNUAL MEETING NEW HORIZONS IN CHEMICAL SENSITIVITIES: STATE OF THE ART DIAGNOSIS AND TREATMENT

The Fungal/Mycotoxin Etiology of Malignancies and Auto-Immune Diseases

The vast majority of malignancies and all of the auto-immune diseases are of unknown cause. Fungi/mycotoxins have been for the most part ignored as documented cause of many malignancies and of auto-immune diseases. The etiopathogenetic mechanisms are not the usual patterns of the invasive-type mycoses nor of mycotoxicoses, but incorporate the occult features of both of these mechanisms. Some of the mycotoxin-induced malignancies are: hepato-cellular carcinoma, esophageal cancer, lung cancer, colon cancer, kidney cancer, breast cancer, colon cancer, endometrial cancer, leukemia, lymphoma, astrocytoma and Kaposi's Sarcoma.

Auto-immune diseases are characterized by the finding of so-called auto-antibodies. It is a most popular concept but biologically fatally defective in that no species of life can make an antibody against itself; particularly causing fatal disease such as scleroderma. Scleroderma is considered to prove the validity of the auto-immune concept with the presence of auto-antibodies. However these are now documented to be antibodies against ubiquitin which is present in many species including fungi. Scleroderma responds well to the antifungal agent griseofulvin. Against whose ubiquitin is the host raising antibodies to, its own, or fungal-derived, in a disease state which responds to an antifungal drug? The auto-immune diseases appearing to have a fungal/mycotoxin origin are: scleroderma, diabetes mellitus, HLA-related disease, rheumatoid arthritis, Sjogren's syndrome, psoriasis & systemic lupus erythematosus. All of the drugs effective in the treatment of these diseases possess antifungal or anti-mycotoxin activity. This includes all NSAIDs.

Fungal/Mycotoxin Etiology of Gout & Hyperuricemia

Gout and hyperuricemia are clinical entities of previously unknown etiology. Fungi/mycotoxins have been ignored as documented cause of both entities. The etiopathogenetic mechanisms are not the usual patterns of invasive-type mycoses nor of mycotoxicoses, but incorporate occult features of both of these mechanisms resulting in abnormal biochemical findings associated with specific granuloma tissue lesions. All of the biochemical findings in gout/hyperuricemia are explainable by fungal production of preformed urates/urate crystals, oxalate, glutamate, glycosaminoglycan, glycoprotein & hormones. Mycotoxins cause hyperuricemia and hyperlipidemia. The gouty tophaceous lesion is a granuloma of the delayed hypersensitivity type and is identical to fungal granulomas. Asteroid bodies, characteristic of fungal lesions, are found in the giant cells in both avian & human gouty tophi. Asteroids are fungal cells coated with fungal antigen+host antibody. Spherules and branching filaments present in tophi have been mis-identified as urates on silver stain which also stains fungal forms the same identical color. Periodic acid Schiff stain has demonstrated faint-staining fungal spherules in gouty lesions. The clinical course of an acute attack of gout is that of a fungal infection with prodrome, all the usual signs of infection, ascending lymphangitis, fever, chills, increase in sedimentation rate, desquamation of the overlying skin. Gout responds to griseofulvin, an antifungal antibiotic which has the same mode of action as colchicine. These clues led to the observation that all drugs and dietary factors improving gout/hyperuricemia possess antifungal and/or anti-mycotoxin activity. The fungal etiology of gout/hyperuricemia provides a rational basis for preventive measures and correct therapy.

The Fungal/Mycotoxin Etiology of Atherosclerosis and Hyperlipldemia

Atherosclerosis and hyperlipidemia are clinical entities of previously unknown etiology. Fungi & their toxins have been ignored as documented etiology of both entities. Hyperlipidemia is induced by a number of mycotoxins. Seasonal variations in hyperlipidemia correlates to seasons of maximal fungal growth and mycotoxin production. It will be shown in this presentation that hyperlipidemia is a protective-toxin binding mechanism that is seen in a number of complex infections and returns to normal with antibiotic therapy and/or toxin bind-agents including charcoal. Atherosclerotic lesions are characterized by lipid deposition, foam cells, endothelial cell damage, smooth muscle cell proliferation, activation of all of the cellular and humoral elements of delayed hypersensitivity, and fibrosis/calcifications. All of these lesions are induced in animals and humans by fungi/mycotoxins. Cyclosporine, a mycotoxin (an immuno-toxic fungal antibiotic) causes accelerated atherosclerosis & hyperlipidemia in the vast majority of transplant patients. Primates developed hyperlipidemia and atherosclerosis when fed Fusarium toxins (corn). Hyperlipidemia associated with lipid-containing vascular lesions are found in sheep ingesting the mycotoxin sporidesmin. In humans, ergots induce spasm, stenosis and/or thrombosis of the coronary, carotid, aortic, renal, and peripheral arteries. Ergot-induced entities include angina, myocardial infarction, arrhythmia, carotid artery occlusion, stroke, intermittent claudication & gangrene. All drugs and dietary measures effective in treating atherosclerosis and/or hyperlipidemia share only antifungal or anti-toxicity activity (lovastatin, griseofulvin, ketoconazole, neomycin, fibrates, etc.).

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