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Abstract for a presentation on:

Cell-mediated Immunity (CMI) in Syphilis in the Context of HIV Infection

The association between HIV and syphilis is overwhelming, yet it seems to be so absent in AIDS. Humoural tests, both treponemal and non-treponemal, are a bonus for the diagnosis of syphilis and are the end result of a successful resistance (CMI response). Sensitivity for the diagnosis based on clinical presentation has always been low. The answer to syphilis diagnosis in persons with HIV infection, or other immune dysfunction, may well rest only in an assay which would show prior sensitization of the lymphocytes. Animal models tend to be anamnestic humourally when reinfected with *T-pallidum*, whereas, generally, humans are not. The only time that human hosts seem to react appropriately to reinfection is when they have been cured of the first infection. There is very little natural immunity to syphilis because CMI in humans has a very hard time getting the upper hand and putting the patient into early latency. This is why the infectious stage is so long. The development of effective CMI to syphilis in the human host is a very precarious affair, while in the animal models we use, this is not the case. Spontaneous cure, as defined in such historical studies as Oslo, would today be considered latency. Undoubtedly those individuals would have positive treponemal tests and effective lymphocyte blast formation in the presence of treponemal antigen, *i.e.*, naturally infected and 'immunized.' The life-style during the twenty years before AIDS emerged was characterized by undoubted multiple (un-diagnosed) treponemal infections and fractionation of developing CMI by sub-optimal therapies. Under-treatment was always considered far worse than no treatment at all, and induced what was termed 'malignant syphilis', the ushering in of later sequellae without the benefit of protection of early exanthem. It is proposed that the inoculation of the virulent treponemes of an early syphilis into an individual in his/her precarious latency will induce AIDS-like immunology. It was always recognized that confounding infections also increased the risk of ineffectual CMI to syphilis. The classical dermatology literature, for instance, demonstrates an abundance of case-definition AIDS in today's risk groups. It is therefore hypothesized that sub-optimal treatment and superinfection in syphilis has historically been a major contributor to this morbidity and mortality from other infections. Early syphilis is an acute infectious disease and very susceptible to beta-lactam drugs, while later syphilis is an auto-immune disease, when symptomatic, and is essentially treated by the host's immune competence, critically dependent on the CMI.