

The Consequences of HIV Denialism

Over the years I have often had discussions with my patients regarding the role of HIV in the progression to AIDS. In the mid 80's and early 90's this happened with moderate frequency and slowly declined over the years as the evidence was increasingly accumulating. Some patients; however, continued to have strong beliefs regarding the role of HIV in causing AIDS. This is a history of one discordant couple with a particularly tragic outcome.

Patient A was an HIV-positive man diagnosed in March 1987. He had remained asymptomatic for many years. We often had long discussions regarding the relevance of HIV to progression to AIDS. He was a strong believer that HIV was not a causative factor in the progression to AIDS. He felt that his own stability with respect to his health was proof positive that HIV had no impact on the likelihood of him progressing to AIDS. In 1992 Patient A met a 31-year-old HIV-negative man.

Patient B, who was also a patient of mine, had been tested for HIV and had been negative in October 1990. He did not do regular testing as he had never had unprotected anal sex and was extremely cautious regarding safer sex. Patient B had been encouraged by his partner to have unprotected sex. The first and only time this happened was in late 1994.

Patient B presented in January of 1995 with the 10 day history of fevers, sore throat, upset stomach, nausea, watery diarrhea, lack of appetite, sweats, and a generalized rash. He was investigated and found negative for syphilis and cytomegalovirus. He was found to be p24 antigen positive and HIV RNA was found to be positive as well. Antibody testing for HIV was found to be negative. He was diagnosed as having an acute seroconversion reaction compatible with HIV and he was treated supportively. Helper CD4 count was 240 on March 24, 1995. He continued to be unwell and was hospitalized on March 30, 1995 when he was diagnosed with *Pneumocystis carinii* pneumonia and esophageal cytomegalovirus. He received treatment at our tertiary care center with expertise in treating HIV. Over the ensuing months he continued to remain clinically ill and he subsequently died on October 6, 1995, 10 months after his initial presentation. Autopsy showed CMV esophagitis and *Pneumocystis carinii* pneumonia.

The question became one of determining whether he had been infected with HIV longer than the acute seroconversion illness would have led one to believe, if he had a more virulent strain of the virus, or whether he had an inherently decreased ability to respond to the virus.

Testing on samples from both Patient A and Patient B was carried out by Dr. Sharon Cassol and Dr. Nelson Michael and their colleagues at the Walter Reed Army Institute of Research in Washington DC. HIV samples from both patients were sequenced. The env genes were so similar, when compared to control sequences, as to establish a transmission link between these two patients. In effect, the two patients were infected with the same virus, and Patient B had been infected by Patient A. The virus transmitted to Patient B was also shown not to be inherently more virulent than the one infecting

Patient A. This work was published in the peer-reviewed scientific literature (Michael NL, Brown AE, Voigt RF, Frankel SS, Mascola JR, Brothers KS, Louder M, Birx DL, Cassol SA. Rapid disease progression without seroconversion following primary human immunodeficiency virus type 1 infection--evidence for highly susceptible human hosts. J Infect Dis. 1997;175:1352-9).

What was found to be different were the abilities of the two individuals to mount an immune response to HIV. Patient B had undetectable humoral and cellular responses to HIV in spite of his being able to mount an adequate immune response when immunized against hepatitis B. Patient A on the other hand had a strong host immune response. This indicated that although the two viruses were identical, the response to the infection of HIV was remarkably different and had significantly different rates of progression. Patient B died within 10 months of exposure to HIV, and Patient A remains well to this day.

The consequences of HIV denialism were profound for Patient A. He had been a strong believer in the lack of connection between HIV and AIDS, and had acted on those beliefs in having unprotected sex with his partner. He had acquired these beliefs because he had heard of the views expressed by Professor Peter Duesberg and had read various of his writings. He felt convinced that he could have unprotected sex because HIV was harmless. As a result, Patient A was initially unwilling to believe that he had done anything that would harm his partner. Once all the testing and evidence was accumulated on the cause of his partner's death, Patient A made the comment "I caused his death." The impact on Patient B was fatal, but the impact on Patient A was likewise profound. He had accepted arguments that HIV was not the cause of AIDS and had lost his partner as a result. His sense of guilt and remorse was deep and took him many years to come to terms with the results of the beliefs that had failed him. I have no doubt that HIV denialism kills people. I saw it happen in a very personal and direct way. I hope it never happens again, but I fear it will until something happens to prevent the further dissemination of lies and distortions about HIV and AIDS.

A handwritten signature in black ink that reads "Robert Voigt". The signature is written in a cursive style with a large, sweeping initial 'R'.

Robert Voigt, BSc, MD