Lymphology 21 (1988) 44

PANEL DISCUSSION: HIV, LYMPH NODES, AND LYMPHOCYTES

DR. M. WITTE: Perhaps we could start with questions of the presenters.
DR. GOTTLIEB: I was impressed by these very beautiful histopathologic demonstrations, but I wonder if you could give us a little more information. First, are these frozen sections or can they be done after formalin fixation?
DR. TENNER-RACZ: No, it must be frozen.
DR. GOTTLIEB: The second point that puzzles me a bit concerns germinal centers; you suggested that viral antigen was there and stimulating B cell function. In view of the lack of CD4 cells in the germinal centers, (a) what would your comment be on that and (b) do you have any evidence that you get specific antibody production to any particular viral antigen?
DR. TENNER-RACZ: Do you mean inside or around germinal centers, are there anti-HIV antibody-producing plasma cells? We assume but couldn't prove that because we have no method to be sure in which particular plasma cell specific antibody is produced. There aren't a large amount of plasma cells, sometimes inside and around the germinal center.
DR. RACZ: A number of CD4 cells, about 25% of the whole cell population, is in the germinal center.
DR. GOTTLIEB: Even in these patients?
DR. RACZ: Yes.
DR. DORFMAN: There have been at least two publications that I'm aware of suggesting that this phenomenon that has been called follicle lysis is not a specific one--it can be found under different circumstances both in tonsil and lymph nodes. To some extent, these papers have suggested that too much is being made of the observations that have been presented this morning. I personally think that is not correct. You've observed very convincing evidence that while these changes may not necessarily be specific, they are quite characteristic. In any patient who presents with generalized lymphadenopathy and fever and in whom the biopsy shows the changes that have been shown, there must be a high rate of suspicion for at least AIDS-related complex if not the AIDS syndrome.
DR. DIEBOLD: I thank you for these comments, Dr. Dorfman, because I agree with you. No observations we showed this morning, Dr. Racz or I, are specific. That's true because we can see all types of lesions in other types of infection. But, the intensity of this lesion and the association of the lesion is highly characteristic of HIV infection. For example, you can see follicle lysis in toxoplasmosis but in this lymph node only a few germinal centers fit exactly into this pattern while it's very impressive in HIV disease. None of these observations in lymph nodes are specific but the association and intensity of the lesion is highly suggestive of HIV infection. Our poster this afternoon illustrates the reliability of histopathology in HIV infection.
DR. WITTE: A question to Dr. Racz. You described impressively dilated lymphatics and many lymphocytes in the subcapsular sinus, but is it really fair to say increased lymph flow because these dilated lymphatics could be obstructed and dilated because of lymphostasis rather than overproduction of lymph. I could conceive easily that early in the disease in ARC, there might be dilatation and increased lymph flow but later, dilatation from obstructed lymph flow could supersede and ultimately perhaps even obliteration of the lymphatics with fibrosis.
DR. RACZ: I agree. What I would like to emphasize is that very rich cell populations are transported through the lymph including great numbers of activated lymphocytes; this is direct evidence of infected cells there. If lymphatic flow is slowed or stopped, the infection might not spread further. So, this is a really new and important question of pathogenesis the background evidence for which we have extensively examined.
DR. ZIEGLER: Do I understand that the Langerhans cells from the skin are found in the sinuses of the lymph nodes, which would suggest that this is a mode of traffic of bringing HIV infection from a broken area of skin, for example, into the lymphatic system? Is that correct?
DR. RACZ: In the first phase of the infection, we see increased numbers of Leu-6 cells which are interestingly focally accumulated in one area of the lymph node. There are other examinations done by Stingl and his group showing some infected Langerhans cells in the skin. Popovic also identified infected macrophages from the skin, and we found Langerhans cells showing positivity with p24 in the skin and also migrating Langerhans cells in the lymphatics of the skin. All this evidence together supports the concept that infected Langerhans cells migrate from the skin into the lymph node. How extensive this transport is we don't know.
DR. WITTE: Dr. Olszewski has examined the transport of Langerhans cells through the afferent lymph--in human and also in experimental situations.