Severe herpesvirus infection beats adult T-cell leukemia/lymphoma

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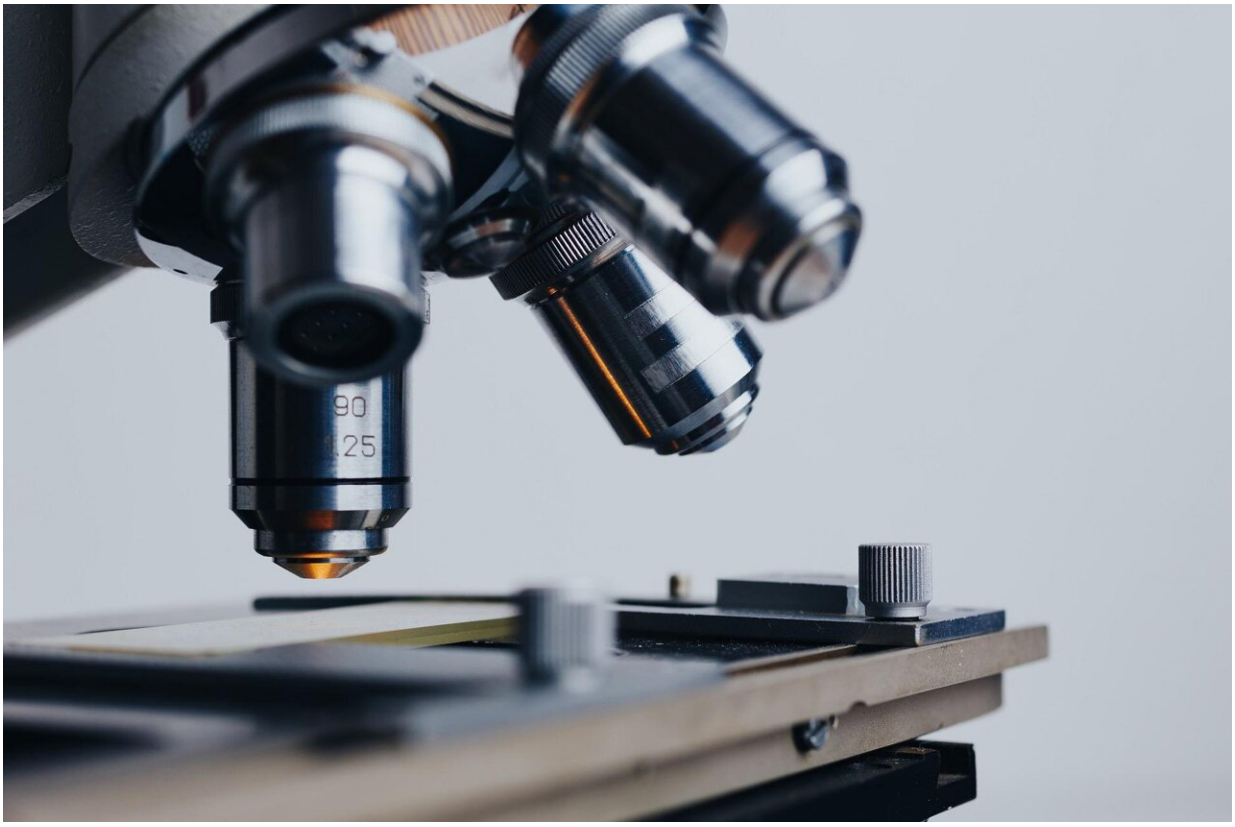

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In a recently published editorial in the journal *Genes & Cancer*, researcher Tatsuro Jo from the Japanese Red Cross Nagasaki Genbaku Hospital's Department of Hematology discussed aggressive type adult T-

cell leukemia/lymphoma (ATLL). ATLL caused by human T-cell lymphotropic virus type 1 (HTLV-1) infection is associated with dismal survival prospects, even after the approval of mogamulizumab (a monoclonal antibody for C-C chemokine receptor 4 antigen).

A large number of genetic and epigenetic abnormalities resulting from the pleotropic effects of HTLV-1 Tax and HBZ reportedly are present in ATLL patients, which may be the cause of their poor response to intensive chemotherapies.

The HTLV-1 sequence is completely distinct from the human genome, making the HTLV-1 gene products highly immunogenic to humans and targets for the humoral and cellular immunity. Actually, antibodies against the HTLV-1 gag and env gene products are ubiquitously detected in HTLV-1 carriers. Furthermore, cytotoxic T lymphocytes (CTLs) against HTLV-1 Tax (TaxCTLs) are often observed in long-term survivors with ATLL treated with or without allogeneic hematopoietic stem cell transplantation. Thus, the activation of antitumor cellular immunity may play an important role among long-term survivors with ATLL, an intractable disease.

In patients with ATLL, the CD4/CD8 ratio in T lymphocytes is often inverted due to a decrease in CD4-positive T lymphocytes, including regulatory T lymphocytes (Treg), after treatment with mogamulizumab. Conversely, we have noticed that the CD4/CD8 ratio is almost always inverted in long-term surviving ATLL patients in the pre-Mogamulizumab era.

Furthermore, several of these patients developed herpesvirus infections, such as herpes zoster and herpes encephalitis, while their disease was controlled by chemotherapy and subsequently exhibited CD4/CD8 ratio reversal. Herpesvirus infection has been reported to strongly activate host cellular immunity.

"Although contracting herpes simplex or herpes zoster is unpleasant, the mechanism by which these herpesvirus infections can produce a therapeutic effect on refractory ATLL via the activation of the host's cellular immunity is extremely interesting and worth further study," says Jo.

More information: Tatsuro Jo, Severe herpesvirus infection beats adult T-cell leukemia/lymphoma, *Genes & Cancer* (2023). DOI: 10.18632/genesandcancer.228. www.genesandcancer.com/archive/v14/

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