Antibody Therapy to Limit the Spread of Ebola Virus

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Antibody therapy for viral infections in the form of immunoglobulin G (IVIG) has been successfully used over the past century [1,2]. IVIG is a major source for antibody therapy due to the diverse repertoire of antibodies against bacterial and viral infections that it contains.

We and others used IVIG for the prophylactic and therapeutic treatment of mice infected with West Nile virus (WNV) and showed that pooled IVIG from donors protected the mice from the development of a fatal WNV disease [3]. No protection of WNV-infected mice was obtained when they were treated with pooled IVIG prepared from American donors in the pre-WNV era. The protective efficacy of the pooled Israeli IVIG preparation was attributed to the fact that WNV is endemic in Israel and many people have been exposed to the virus or even suffered from mild infections. The efficacy was improved with the treatment of West Nile hyperimmune IVIG (WIVIG). The success of these treatments was time and dose dependent. It was noted that prophylactic therapy was always better than the therapeutic treatment [4].

There is good evidence that antibody therapy can be effective in protecting against Ebola infection; nonetheless, as in other diseases it is probably also time and dose dependent [5]. Since in most cases exposure to Ebola patients can be detected before clinical symptoms appear, it may be more efficacious to treat these human contacts – including health workers – in addition to the patients, with hyperimmune Ebola serum or IVIG prepared from recovered individuals.

In order to control the pandemic, prophylactic treatment should be given. That is, prevention should become the central strategy, and IVIG prepared from convalescent patients should be used at earlier stages before clinical symptoms emerge.

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References

Capsule

Cancer cell-autonomous contribution of type I interferon signaling to the efficacy of chemotherapy

Some of the anti-neoplastic effects of anthracyclines in mice originate from the induction of innate and T cell-mediated anticancer immune responses. Sistigu et al. have demonstrated that anthracyclines stimulate the rapid production of type I interferons (IFNs) by malignant cells after activation of the endosomal pattern recognition receptor Toll-like receptor 3 (TLR3). By binding to IFNα and IFNβ receptors (IFNARs) on neoplastic cells, type I IFNs trigger autocrine and paracrine circuitries that result in the release of chemokines (C-X-C motif) ligand 10 (CXCL10). Tumors lacking Tlr3 or Ifnar failed to respond to chemotherapy unless type I IFN or Cxcl10, respectively, was artificially supplied. Moreover, a type I IFN-related signature predicted clinical responses to anthracycline-based chemotherapy in several independent cohorts of patients with breast carcinoma characterized by poor prognosis. These data suggest that anthracycline-mediated immune responses mimic those induced by viral pathogens. The authors surmise that such ‘viral mimicry’ constitutes a hallmark of successful chemotherapy.

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