

Cancer chemotherapy-induced lymphocytosis: a revolutionary discovery in the medical oncology.

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The recent advances in the investigation of tumor immunobiology have suggested that cancer chemotherapy, in addition to its well known cytotoxic activity, may play modulatory effects on the endogenous production of cytokines involved in the control of both tumor angiogenesis and antitumor immunity.

Cancer chemotherapy constantly acts with inhibitory effects on anti-bacterial, anti-viral and anti- mycotic immune responses, whereas its action on anticancer immunity, which is mainly mediated by lymphocytes, has still to be better investigated and defined. The present study was carried out to evaluate the influence of chemotherapy on lymphocyte count and its relation to the clinical response in cancer patients suffering from the most commonly frequent tumor histotypes, including lung, colorectal, breast and prostate carcinomas.

The study included 144 consecutive metastatic solid tumor patients. Lung cancer patients were treated with cisplatin plus gemcitabine, colorectal cancer patients received oxaliplatin plus 5-fluorouracil, while those affected by breast cancer or prostate carcinoma were treated with taxotere alone. An objective tumor regression was achieved in 66 out of 144 (46 percent) patients, whereas the remaining 78 patients had only a stable disease (SD) or a progressive disease. Independently of tumor histotype and chemotherapeutic regimen, a lymphocytosis occurred in patients who achieved an objective tumor regression in response to chemotherapy, and lymphocyte mean count observed at the end of the chemotherapeutic treatment was significantly higher with respect to the values seen before the onset of treatment.

On the contrary, lymphocyte mean number decreased on chemotherapy in patients with SD or PD, even though the decline was statistically significant with respect to the pretreatment values in the only patients who had a PD in response to chemotherapy.

This study would suggest that chemotherapy itself may paradoxically act, at least in part, as a cancer immunotherapy by inducing lymphocytosis, as well as previously demonstrated for the only immunotherapy with IL-2, probably by modulating the cytokine network and correcting the altered endogenous production of cytokines, responsible for cancer-related immunodeficiency.