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Serum leptin levels during cancer chemotherapy

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Dear Sir,

Cancer chemotherapy induces anorexia by stimulation of serotonin, dopamine, histamine, and acetylcholine secretion [2]. Leptin, the adipocyte-specific product of the *ob* gene, regulates food intake [3, 4]. We measured serum leptin levels in 27 nondiabetic patients (12 men and 15 women, aged 19–83 years) who received chemotherapy for hematological malignancies (13 with acute myelogenous leukemia, one with multiple myeloma, and 13 with malignant lymphoma). Chemotherapy consisted of enocitabine, daunorubicin, mercaptopurine, and prednisolone for leukemia, melphalan and prednisolone for myeloma. Cyclophosphamide, doxorubicin, vincristine, and prednisolone (CHOP regimen) were given to all the patients with lymphoma except one who received the ESHAP regimen (Fig. 2). After we had received informed consent, blood samples were obtained by venipuncture at 10:00 a.m. 1 day before, during, and 2 weeks after the completion of chemotherapy. Serum leptin levels were determined using the Human Leptin RIA kit (LINCO Research, St. Charles, Mo., USA). As shown in Fig. 1, serum leptin levels were significantly elevated during chemotherapy (mean \pm S.E.: 8.22 ± 0.91 ng/ml) compared with those before and after chemotherapy (5.30 ± 0.68 ng/ml and 5.27 ± 0.63 ng/ml; $p = 0.008$ and 0.0142 , respectively; paired t -test). In contrast, when the food intake rate is designated as the portion of hospital food eaten, in order to measure appetite, the rates during chemotherapy ($69 \pm 6.4\%$) were lower than those before and after it (75 ± 6.0 and $72 \pm 5.8\%$, respectively). However, there was no statistically significant difference. Body mass indices prior to

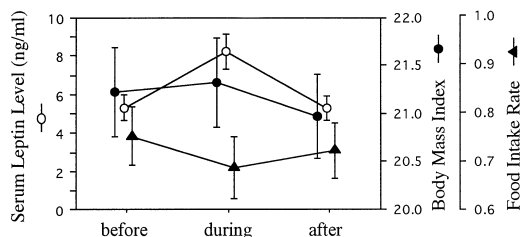


Fig. 1. Serum leptin levels, food intake rates, and body mass indices before, during, and after cancer chemotherapy. Bars represent S.E. for 26 patients

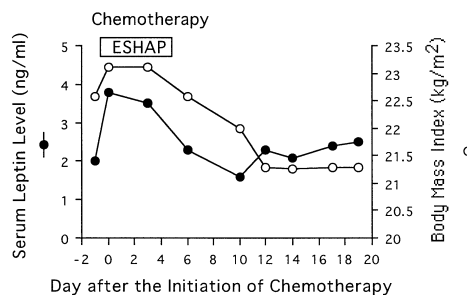


Fig. 2. Serial changes in serum leptin levels and body mass indices. The patient with non-Hodgkin's lymphoma received chemotherapy composed of cisplatin, etoposide, cytarabine, and methylprednisolone (ESHAP regimen). Food intake rates were kept 1.0 throughout the entire period

chemotherapy (21.23 ± 0.46 kg/m²) were not significantly different from those during it (21.33 ± 0.47 kg/m²), and they decreased significantly after chemotherapy (20.98 ± 0.44 kg/m²) compared with during it ($p = 0.0155$), probably due to anorexia induced by the chemotherapy. There was no significant difference in leptin levels when the chemotherapeutic regimens were compared.

Serial changes in the serum leptin level were examined during the course of cytoreductive therapy in one patient with non-Hodgkin's lymphoma (Fig. 2). The

leptin level increased during chemotherapy, and then dropped to the pre-chemotherapy level. The body mass indices fell gradually after chemotherapy.

Serum leptin levels increased inversely as appetite decreased during chemotherapy. Observed elevation of serum leptin levels during chemotherapy was not as high as levels reported in obese human beings [1]. This may be because chemotherapeutic agents in this study were mildly emetogenic drugs. Our observation suggests the possibility that chemotherapy by itself induces leptin overproduction, which is then responsible for suppression of hunger and therefore for weight loss.

References

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