



Influence of anticancer therapy on oxidation phenotype and acetylation phenotype in patients with acute myeloblastic leukemia

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Abstract:

The aim of this study was to determine whether antineoplastic cytostatic therapy induces changes in the oxidation or acetylation phenotypes in patients with acute myeloblastic leukemia (AML).

The investigations involved 22 patients with AML undergoing chemotherapy with daunorubicin, cytosine arabinoside, etoposide and mitoxantrone. The oxidation phenotype prior to therapy and after termination of induction was examined in all 22 patients and was examined in 10 patients after termination of the first consolidation cycle. The acetylation phenotype was examined prior to therapy and after termination of induction in 21 patients and after termination of the first remission consolidation cycle in 9 patients. The oxidation phenotype was determined by means of the method by Eichelbaum and Gross. The acetylation phenotype was determined using Varley's modification of the Bratton-Marshall method.

Anticancer therapy affected the oxidation phenotype, causing decreased activity of the cytochrome P450 isoenzyme CYP2D6. This decrease suggests that daunorubicin, cytosine arabinoside, etoposide and mitoxantrone may impair the metabolism of other active substances metabolized by this isoenzyme, which should be taken into consideration in planning the dosage scheme in individual patients and considering interactions between drugs. Evaluation of the effect of administered cytostatic drugs on acetylation phenotype revealed no statistically significant decrease in the rate of sulfadimidine acetylation.

Key words:

acute myeloblastic leukemia, AML, oxidation phenotype, acetylation phenotype, genetic polymorphism, anticancer therapy
