Letters and correspondence submitted for possible publication must be identified as such. Text length must not exceed 500 words and five bibliographic references. A single concise figure or table may be included if it is essential to support the communication. Letters not typed double-spaced will not be considered for publication. Letters not meeting these specifications will not be returned to authors. Letters to the Editor are utilized to communicate a single novel observation or finding. Correspondence is to be used to supplement or constructively comment on the contents of a publication in the journal and cannot exceed the restrictions for Letters to the Editor. The Editor reserves the right to shorten text, delete objectional comments, and make other changes to comply with the style of the journal. Permission for publication must be appended as a postscript. Submissions must be sent to Paul A. Chervenick, M.D., Associate Editor; American Journal of Hematology, H. Lee Moffitt Cancer Center, 12902 Magnolia Drive, Suite 3157, Tampa, FL 33612-9497 to permit rapid consideration for publication.

Retreatment With Low-Dose Cytarabine in Patients With Previous Central Nervous System Toxicity

To the Editor: During the last 10 years, due to high-dose regimens and the treatment of more elderly patients, numerous reports of central nervous system (CNS) toxicity secondary to cytarabine therapy have been published. There is little information about the physiopathology of CNS toxicity, but it seems to be dose-dependent and related to age and previous CNS toxicities [1–5]. With regard to this last feature, there are no reports in the literature that analyze retreatment, with lower doses of cytarabine of patients who have suffered previous CNS toxicity.

A 55-year-old male was diagnosed to have myelodysplastic syndrome 1 year before he transformed to an acute non-lymphoblastic leukemia (AML-M7 of the French-American-British classification). He was treated with a 3 + 7 regimen of cytarabine (200 mg/m²), continuous infusion daily over 7 days, and daunorubicin (45 mg/m²) by endovenous bolus on the first 3 days. He developed chemotherapy-induced aplasia over 14 days, and then relapsed with 60% of blasts in bone marrow. He received high-dose cytarabine (2 g/m² every 12 hr for 4 days) and mitoxantrone (10 mg/m² for 3 days) as an intensification regimen.

Four days after cessation of therapy, he developed progressive cerebellar syndrome characterized by severe ataxia, dysmetria, and dysarthria, with mild obnubilation and disorientation. This clinical picture remained unchanged for 72 hr and then slowly resolved in 1 week. CT scanning and lumbar puncture performed early after onset of the first symptoms were negative.

By day 31 he had achieved partial remission, with 10% of leukemic blasts on bone-marrow examination along with mild pancytopeny. He was treated with a salvage protocol which included BCNU (200 mg/m², day 1), VP-16 (300 mg/m², days 2 and 3), ansamycin (100 mg/m², days 1–3), and cytarabine (300 mg/m², continuous infusion over days 1–4). Tolerance was excellent, and the patient developed no neurologic toxicity. He achieved a complete remission and remains alive and event-free 3 months after cessation of therapy.

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References

Rare β-Thalassemia Mutation IVS-II-848 (C-A) First Reported in a Turkish Cypriot Family

To the Editor: Until now about 180 different mutations, affecting many different processes in globin gene expression, have been reported as causes of β-thalassemia [1]. Knowledge of the geographic or ethnic distribution of these mutations facilitates effective prenatal diagnosis programs [2–5].
population has primarily four unique β-thalassemia alleles: IVS-I-110 (G-A), IVS-I-6 (T-C), IVS-I-1 (G-A), and IVS-II-745 (C-G), accounting for 96% of the mutations in this ethnic group. Other alleles are rare, which is indicative of the high degree of genetic homogeneity in this population.

Recently we identified a 25-year-old Turkish Cypriot woman whose hematological analysis revealed mild anemia (HbA, 11.2 g/dl) and hypochromia (MCV, 69 mm; MCH, 21.3 pg). A diagnosis of β-thalassemia carrier status was made, later shown to be inherited from her clinically asymptomatic father. DNA sequencing revealed a C-A substitution at nt 848 of the IVS-2 region (Fig. 1). This was confirmed by allele-specific oligonucleotide hybridization. This mutation was first discovered in an American Black population, but has an estimated frequency of 11% in Egyptians [6]. The mutation abolishes the 3’ consensus sequence and leads to aberrant RNA processing with a resultant β+ phenotype. The source of mutations in the Turkish Cypriot population is not known, but current haplotype analysis may clarify this.

**SÜRKÜ TUZMEN**  
A. NAZLI BASAK  
Department of Molecular Biology and Genetics, Bogazici University, Istanbul, Turkey

**EROL BAYSAL**  
Department of Biochemistry and Molecular Biology, Medical College of Georgia, Augusta, Georgia

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