

studied by classifying complex rearrangements and detecting cryptic abnormalities overlooked by standard cytogenetics. In addition, SKY analysis resulted to be a relevant tool in disclosing the origin and mapping progression of disease in four of the five patients revealing AML specific gene and breakpoint rearrangements.

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## Acute leukemia in Jehovah's Witnesses: a challenge for hematologists

With the aim of demonstrating the possibility of treating acute leukemia in Jehovah's Witnesses by protocols which include a marrow transplantation procedure without need of transfusions, we report our experience on 5 patients. Three patients had acute lymphocytic leukemia and two acute myeloid leukemia, two received autologous bone marrow transplants and one bone marrow transplantation without transfusions. One died because of anemia during induction. Our conclusion is that acute leukemia in Jehovah's Witnesses can be treated with protocols employed in transfused patients.

Sir,

Jehovah's Witnesses object to transfusions of blood products; thus, the management of acute leukemia in these patients is of great difficulty. Reports emphasize the lower rate of remissions and cure of Jehovah's Witnesses with acute leukemia, because of the reduced cytotoxicity of the regimens adopted in these patients.<sup>1,2</sup> Here we report on five recent cases of Jehovah's Witnesses with acute leukemia, trying to give an example that could be useful for physicians faced with a similar challenge. Table 1 summarizes the patients' data.

Case #1 was an 11-year old boy admitted to hospital October 1998 because of hyperleukocytosis and T-cell acute lymphocytic leukemia (T-ALL) ALL-T (blast cells  $320 \times 10^3/\mu\text{L}$ ). His hemoglobin was normal. He received induction chemotherapy according to the AIEOP 9503 protocol. Hematologic recovery occurred day +30 without him needing transfusions. Because of persistence of blasts he received a bone marrow transplant (BMT) from his brother with chemotherapy (CTX) and fractionated total body irradiation (TBI) as conditioning treatment without transfusions; he relapsed and died eight months following BMT.

Case #2 was a 24-year old man with acute myeloid leukemia (AML)-M2; he received a combination of idarubicin (10 mg/m<sup>2</sup>) fludarabine (25 mg/m<sup>2</sup> days 1 to 5) and aracytin (2 g/m<sup>2</sup> days 1 to 5). Erythropoietin was started on day 5 but his hemoglobin dropped to 2.7 g/dL and the platelets became uncountable. Nevertheless, the patient recovered from cytopenia around day +20 and achieved CR. After a second course of therapy, he underwent autologous PBSC with busulfan 12 mg/kg and melphalan 120 mg/m<sup>2</sup> as conditioning therapy. He relapsed and died

**Table 1. Jehovah's Witnesses with acute leukemia.**

| N°Age/Sex (years) | FAB     | Therapy | BMT/ABMT | Result | Status | Follow-up |
|-------------------|---------|---------|----------|--------|--------|-----------|
| 1 11/M            | ALL-L2T | AIEOP   | BMT      | PR     | Dead   | 8 months  |
| 2 23/M            | AML-M4  | FLAI-5  | ABMT     | CR     | Dead   | 9 months  |
| 3 17/F            | AML-M3  | AIDA    | None     | CR     | CR     | 14 months |
| 4 30/M            | ALL-L3  | Magrath | ABMT     | CR     | CR     | 12 months |
| 5 44/F            | ALL-L2  | GIMEMA  | None     | NE     | Dead   | 6 days    |

CR=complete remission, PR=partial remission, NE: not eval

nine months after transplantation.

Case #3 was a 17-year old girl who presented in June 1999 with AML-M3. We modified the schedule of AIDA by reducing, at the beginning, the number of doses of idarubicin which were rescued later. She obtained hematologic and molecular remission without platelet support and now, following consolidation, is under maintenance therapy.

Case #4 was 30-year old man diagnosed as having ALL-L3 in September 1999. He received Magrath's protocol for Burkitt's type lymphoma. He obtained remission eighteen days following the start of therapy with a drop of hemoglobin to 5.9 g/dL. With erythropoietin administration his hemoglobin level rapidly recovered and he completed all four phases of treatment and received an autologous PBSC as consolidating treatment. At no time did he have any transfusions of blood products.

Case #5 was a 44-year old woman with ALL-L2; because of a delay of one month her hemoglobin level was 5.4 g/dL at the time therapy was started: she died because of anemia 6 days later.

The treatment of acute leukemia is strongly linked to the possibility of transfusing patients; however we demonstrated that chemotherapy in conventional protocols and allogeneic or autologous BMT could be applied without blood support. Few experiences on successful treatment of acute leukemia without transfusions in Jehovah's Witnesses have been reported.<sup>3,4</sup> A relevant point is that therapy with conventional protocols should be started as soon as possible because one week of treatment and two weeks of marrow aplasia following treatment in order to get remission may be an acceptably short period of time to have a level of Hb still compatible with life before recovery begins. Some patients not transfused during the course of therapy for acute leukemia have the possibility of reaching complete remission with few adjustments of protocols; we are convinced that the possibility of inducing remission without transfusions is easier in patients with ALL than

AML because the myelopoiesis emerges earlier after the beginning of therapy. However a shorter induction therapy than that actually proposed in schemes for AML could be the choice. We conclude that the medical challenge is always, despite the difficulties, to give a chance of cure to patients.

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### Telomerase: obviously activated in the accelerated phase of chronic myeloid leukemia

**We report that the telomerase activity of bone marrow samples from accelerated phase and blast crisis chronic myeloid leukemia (CML) was higher than that in chronic phase CML and control samples. This difference might be due to the number of immature cells, but some inherent changes might also exist during the transformation of CML.**

Sir,

Telomerase is a ribonucleoprotein polymerase that synthesizes telomeric repeats on the 3' ends of eukaryotic linear chromosomes to maintain telomeres.<sup>1</sup> Numerous reports have presented that telomerase activity was expressed in most human tumor cells, but not in normal cells, except germ line cells, hematopoietic stem cells and activated peripheral blood lymphocytes,<sup>2-5</sup> which suggested that the activation of telom-