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Paroxysmal Nocturnal Hemoglobinuria, Exploring the Mystery of an Unknown Art

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About the Study

The development of symptomatic PNH preceding a relapse of AML reveals a complex constellation immunity, clonal expansion, and leukemogenesis [1].

The PNH clone was allowed a massive expansion prior to AML relapse. This phenomenon triggers a few debatable questions. Taking into account the clonal dynamics, the proliferation of an AML clone is much faster than that of an acquired PIGA mutated clone.

This sparks a genuine controversy if the PNH clone originating from the donor cell. It is still unclear if the doner T cell surveillance in a BMT affects differentially the cohabitation and expansion of these two clones.

We were unsuccessful to study the archival material of 2009 of the donor and the recipient because of limited access. It could be secondary leukemia rather than a relapse reasonably hypothesized by Prof Lucio Luzzato in personal communication (Figures 1 and 2).

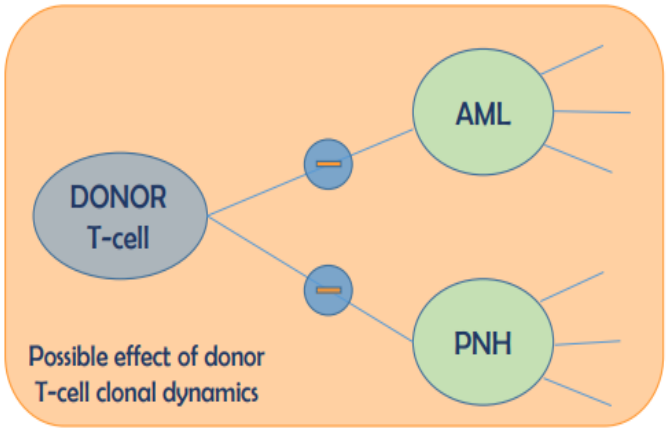

Figure 1. Possible effect of donor T-cell clonal dynamics.

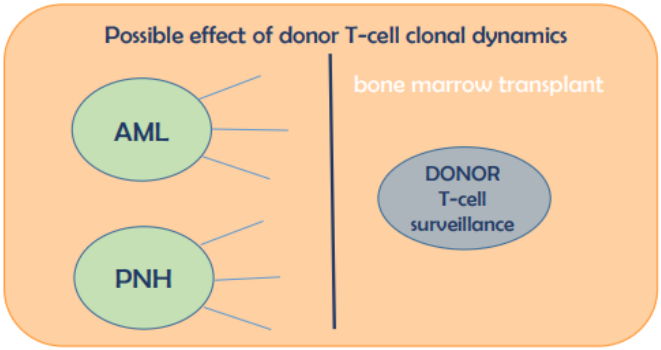

Figure 2. Possible effect of donor T-cell clonal dynamics of bone marrow transplant.

Another plausible hypothesis, concerning the presence of a PNH clone in 2009 might have been overlooked. This could be suppressed due to allogeneic T cells and finally surfaced with clinical relapse or else a secondary PNH different from the original PIGA mutated clone. There is a report to support this hypothesis concerning the emergence of a new somatic PIGA mutation at relapse of following syngeneic bone marrow transplantation for a PNH patient in the pre-complement inhibitor era (Figure 3) [2].

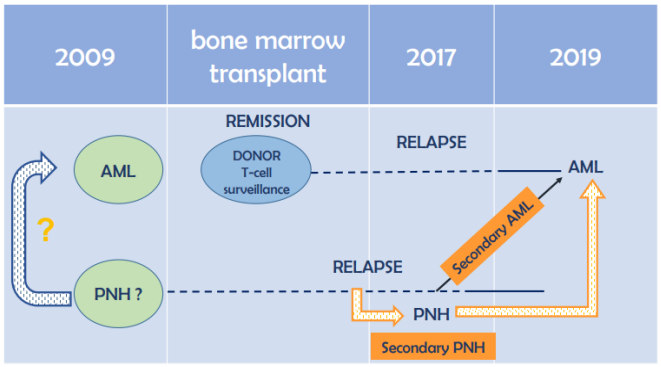

Figure 3. Possible hypothesis of triggering AML and PHN.

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