outcome, but it can also identify potential drug targets. The gene expression data were used as a basis for suggesting using TBXAS1 and SEMA3F as drug targets (96).

AML is treated by two therapeutic steps (97). The first step is induction, which results in remission. The second step is post-remission therapy, which prevents relapse. Drugs in use for treating AML include clofarabine (purine analogue), laromustine (DNA alkylating agent), gemtuzumab ozogamicin (antibody), decitabine (inhibitor of DNA methyltransferase), and azacitidine (inhibitor of DNA methyltransferase) (98).

2. Acute promyelocytic leukemia

APL is a subset of AML, and comprises about 10% of adults with AML (99). APL manifests itself by spontaneous bleeding (100). The bleeding is potentially fatal, and it is recommended that treatment be started, after an emergency consultation with a hematologist, before the diagnosis is confirmed. Death can result by bleeding in the central nervous system, lungs, or gastrointestinal tract (101).

APL involves a chromosomal defect, where there is a translocation between chromosomes 15 and 17. This translocation generates the fusion gene involving the PML gene and retinoic acid receptor-alpha gene (RAR-alpha). PML stands for "promyelocyte." The resulting fusion gene and the expressed fusion protein are called PML-RAR-alpha. The fusion protein blocks the differentiation of the cells (102).

The disease is highly curable, where treatment involves all-trans-retinoic acid (a form of vitamin A) plus anthracycline. Patients also receive transfusions of platelets. APL can also be cured by administering all-trans-retinoic acid plus arsenic trioxide. Arsenic trioxide is As_2O_3. According to Nayak et al. (103) studies on APL patients have shown that all-trans-retinoic acid alone improves survival, arsenic trioxide alone improves survival, and that the combination of both drugs further improves survival, that is, the effects are somewhat additive.

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