From the Editor

In this issue of the *American Journal of Hematology/Oncology,*® Drs Cheminant and Hermine present a clinical, biological, and therapeutic overview of mantle cell lymphoma (MCL), an entity not seen commonly in general oncology practice and one that has a distinct biological driver. MCL was initially described by morphological criteria,

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and it was recognized as having a different clinical course than other types of lymphoma as curative combination chemotherapy regimens for lymphomas evolved. As protein biomarkers, along with cytogenetic and, eventually, molecular tools were used to classify lymphomas, the causative and hallmark feature of MCL, the t(11;14) translocation and resulting cyclin D1 overexpression, was identified. However, this did not explain the spectrum of clinical behavior—including not only more resistant disease and late relapses but also very indolent disease that, in some cases, maintains long remissions. Nevertheless, for a majority of these cases, similar to other aggressive lymphomas, more intensive regimens and autologous transplants are typically recommended. Specific biomarkers and clinical features may allow us to discern those patients who do not even need initial treatment, whereas it remains difficult to reliably identify those who need aggressive treatment, including transplant upfront, because survival differences have not been uniformly seen.

Still, significant progress with induction regimens has been achieved—some that are common across lymphoid malignancies, such as the use of rituximab, and others that have been particularly helpful in older patients or those with comorbidities, such as the use of bendamustine or the proteasome inhibitor bortezomib. The latter is associated with shorter progression-free survival and is more typically used in the salvage setting. The ongoing hazard of recurrence over time has prompted the development of maintenance regimens along with the emerging use of molecular monitoring tools that are detailed in the review. Newer targeted therapies still remain a big hope—even for the highly refractory and aggressive subsets, such as ibrutinib, the Bruton tyrosine kinase inhibitor, or CDK 4/6 inhibitors, which logically target the driver lesion, cyclin D. Despite the relative rarity of this tumor type, trials continue to inform more personalized and successful diagnostic and therapeutic strategies.