others have observed LVEF improvement in selected patients after a longer treatment with chelators other than DFP.2

Table 1 makes it clear that several chelators are available that are effective at clearing liver and cardiac iron, and future studies will help elucidate mechanisms of action. None of these studies solve the overwhelmingly largest problem in the management of transfusional iron overload, which is the fact that patients do not take their medication and that deaths continue to occur because of poor adherence.

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that is, could the hepatic or other amyloid organ involvement be due to a culprit protein unrelated to the clonal light chain? Typing the amyloid, usually by mass spectrometry, is clearly necessary when patients have an MG, a tissue diagnosis of amyloid, and possibly another amyloid-forming protein. This situation may occur in patients with MG who are older men with age-related TTR cardiac amyloid or blacks with hereditary amyloid (4% prevalence of mutated TTR), as well as in patients with inflammatory disorders such as severe gout or inflammatory bowel disease and AA amyloid or in Hispanics or victims of chronic hepatitis C who have hepatic and/or renal amyloid due to LECT2. As pathologists adapt the findings of Mereuta et al to immunohistochemical staining regimens for hepatic and renal biopsy specimens containing amyloid, mass-spectrometry typing may not be required; for now, however, given its ease and reliability, it remains the gold standard for typing amyloid, particularly when there are possibly 2 amyloid-forming proteins present in the same patient.

In the past several decades, much progress has been made in the diagnosis, prognostic evaluation, and treatment of patients with clonal plasma cell neoplasms. In fact, in practice today, we follow the principle that the genetic profile and stage of the clonal plasma cell disease and the pattern of clone-related organ damage are the axes that pose risk to patients. Just as genetically high-risk MM confers the possibilities of resistant relapse and shortened survival, AL-related visceral involvement confers the risks of organ failure and, in symptomatic cardiac patients, of shortened survival. Conversely, it must be noted that treating because of presumed AL in patients with MGUS, smoldering MM, or WM when the amyloid is in fact due to a non-AL protein confers the risk of therapy with no benefit. The advance that Mereuta et al offer us is a reminder of our promise to first do no harm.

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REFERENCES

LECT2 makes the amyloid list

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