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Dear Editor,

We read with great interest the recent paper by Siniscalchi et al. [1] in which they described a 69-year-old woman with a cutaneous relapse of multiple myeloma (MM) after six cycles of MPT regimen (melphalan, prednisone, and thalidomide) and thalidomide maintenance therapy. They reported a complete response after four cycles of bortezomib (B) and dexamethasone, suggesting a possible employment in cutaneous plasmacytomas. We would like to make few comments and share some experience since we recently treated and described [2] a 75-year-old IgG/k multiple myeloma patient resistant to MPT, that after two cycle of B (alone without dexamethasone) at usual doses, despite a very good response in the serum monoclonal component, had a cutaneous localization of extramedullary MM (histologically confirmed). After the addition of dexamethasone to B, our patient showed a complete disappearance of the cutaneous lesions.

Thus, the first comment is that our patient had a MM progression consisting of a cutaneous localization of monoclonal plasmacells during B treatment, which is different to the observations of Siniscalchi et al. This peculiar MM behavior makes the cutaneous tissue as a “sanctuary” not reachable by B. Interestingly, this hypothesis could be also in

part explained by the pharmacokinetics of B [3]: In fact, in preclinical murine studies, radiolabeled [¹⁴C] B levels were higher in most organs than in the whole blood within 1 h, with the exception of the brain, testis, and adipose tissue.

Secondly, in our case, the addition of dexamethasone to B was resolute; thus, it could be that in the case described by Siniscalchi et al., the reason of response was due to the corticosteroid.

Although bortezomib has a great efficacy in soft tissue plasmacytomas, it has rarely been described in cutaneous MM. More patients need to be treated and reported with particular attention to these observations.

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