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Journal Articles

2014

The authors reply

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Recommended Citation

Jhaveri KD, Shah H, Radhakrishnan J. The authors reply. . 2014 Jan 01; 85(1):Article 2248 [p.]. Available from: https://academicworks.medicine.hofstra.edu/publications/2248. Free full text article.

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despite the advent of tyrosine kinase inhibitors,⁴ this complication should be added to future reviews of this rapidly moving field.

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Kidney International (2013) 85, 213-214; doi:10.1038/ki.2013.409

The Authors Reply: Shavit *et al.*¹ and Demoulin and Jadoul² provide additional examples of chemotherapy-induced thrombotic microangiopathy (TMA). We thank these authors for reviewing the possible role of the above chemotherapeutic agents in the development of TMA. Both interferon- α^3 and $-\beta^{4,5}$ have been associated with TMA. However, the mechanism by which interferon- α and - β could induce TMA lesions remains unclear. Drugs such as interferon might disrupt complex pathways of complement regulation and have a role in endothelial damage.³ In recent years, mutations of complement system regulators have been directly implicated in the induction of atypical hemolytic uremic syndrome.⁶ It is possible that alteration of alternate pathway of the complement system might be involved in many such cases of chemotherapy-induced TMA. We thank Shavit and colleagues⁷ to bring to our attention their previously reported case series on renal TMA seen following pegylated liposomal doxorubicin (PLD) use. While PLD was implicated as a cause of the renal TMA in the three cases reported by these authors,⁷ two of the three patients had also received either bevacizumab or gemcitabine. Bevacizumab and gemcitabine, as noted in our review,8 have also been associated with renal TMA. Nevertheless, anthracycline therapy is widely used in many cancer treatments and knowledge of both glomerular epithelial⁹ and endothelial damage⁷ by such agents is useful to the onco-nephrologist. In addition, since the writing of our review,8 other novel chemotherapeutic agents have also been associated with glomerular disease. Sasaki et al.¹⁰ report a case of diffuse proliferative immunoglobulin A-dominant immune complex glomerulonephritis in a patient who received cetuximab for recurrent oral squamous cell carcinoma. In this case, the authors make note of the rapid recovery of kidney function within 3 weeks of discontinuing the drug. This case suggests the need for monitoring kidney function in patients being treated with recently developed monoclonal antibodies. Finally, a recent letter to the editor suggested an association of lenolilomide (anti-multiple myeloma agent) with minimal change disease.¹¹ As the field of oncology is rapidly growing, both oncologists and nephrologists will need to be vigilant for any glomerular nephrotoxicities that might occur following the use of novel chemotherapeutic agents.

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- 11. Jamme M, Galichon P, Hertig A. Minimal change disease and lenalidomide. *Am J Kidney Dis* 2013; **pii: S0272-6386**: 01010-0101.

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Kidney International (2013) 85, 214; doi:10.1038/ki.2013.411

A hip fracture in a dialysis patient with $A\beta 2M$ amyloidosis

To the Editor: We read the case report presented by Bataille *et al.*¹ with great interest. In Japan, the number of patients whose dialysis vintage is more than 25 years reached 12,293 at the end of 2012. Although the number of operative cases of carpal tunnel syndrome is declining, A β 2M amyloidosis is still a common disease among long-term hemodialysis patients. For instance, there are about 100 patients whose