PEGFILGASTIM INDUCES A CRESCENTIC GLOMERULONEPHRITIS

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Hodgkin's Lymphoma (HL) and the Human Immunodeficiency Virus (HIV) have been associated with a variety of glomerulonephritic processes. The following scenario involves a 48 year old HIV male who developed rapid and progressive glomerulonephritis (RPGN) with a biopsy proven crescentic glomerulonephritis (CGN) within one week after he underwent the first chemotherapeutic cylcle to treat his recently diagnosed HL. Prior to the treatment of HL the patient was successfully treated with HAART and his renal function was normal with a GFR of 92 ml/min. Despite a normal renal function, the patient's U/A demonstrated a new onset proteinuria and microscopic hematuria four months prior to the chemotherapy administration.

The chemotherapy regimen included pegfilgastim, dexamethasone, dacarbazine, doxorubicin, and vinblastine. Glomerular macrophage colony-stimulating factor (GM-CSF) has been demonstrated to be a mediator in the pathogenesis of CGN. Filgastim, an analogue of GM-CSF, has been associated with the new onset proteinuria and microscopic hematuria. After the patient received his chemotherapy, he developed leukocytosis, culture-negative fevers, and acute renal failure. This clinical case likely represents the excacerbation of a mild glomerulonephritis into a full blown CGN after pegfilgastim administration.