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Modifications of Renal Function in Cancer Patients Undergoing Repeated and Frequent Administrations of Iodinated Contrast Medium (CM): A Multicentric Retrospective Study from Italy

Laura Cosmai,1 Marta Pirvano,2 Giulia V. Re Sarto,3 Camillo Porta,4 Maurizio Gallieni,1,4 Aziende Socio Sanitarie Territoriali Fatebenefratelli Sacco, Milano, Italy; 1Universita degli Studi di Milano Dipartimento di Scienze Biomediche e Cliniche Luigi Sacco, Milano, Italy.

Background: Contrast-enhanced computed tomography (CECT) is the imaging of choice for the diagnosis, staging, and follow-up of cancer patients, not to take into account its role to evaluate response to oncological treatments; in fact, it has been estimated that 47% of all CECTs are prescribed by Oncologists. Comorbidities, nephrotoxic concomitant medications, such as chemotherapy and other causes (nausea and vomiting, diarrhea, etc.) expose cancer patients to a higher risk of developing acute kidney injury (AKI) from CM. Risk factors, definition (PC-AKI vs CI-AKI) and preventive measures have been recently reconsidered, ultimately downsizing the incidence of this adverse event.

Methods: Aim of this study was to retrospectively assess the effects on renal function of repeated CM administrations in 407 oncological patients on active treatment, collected from 5 Italian oncology departments; patients should have undergone at least 3 CECT (on the average 3.5) within a single year (Fig I).

Results: According to our study, neither significant differences in eGFR values (calculated with the CKD-EPI formula) between the baseline and the different post-CECT timepoints, nor AKI cases (defined according to the RIFLE criteria), were recorded.

Conclusions: Repeated CM administrations in cancer patients did not lead to a worsening of renal function, confirming that CI-AKI has a significantly lower incidence than previously thought. Notably, 80% of the patients examined were found to be at low-risk, highlighting some kind of reluctance of Medical Oncologists and Radiotherapists to perform CECTs in these patients. On the contrary, the administration of CM could, and should, be freely used in cancer patients, even in those at a higher risk.

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Proliferative Glomerulonephritis with Monoclonal Immunoglobulin Deposits: Searching for the Underlying Clone

Vincent Lavagne1,2,3,4,5 Sébastien Bender,1 Jean-Michel Goujon,1 Guy Touchaud,1 Christophe Sirac,2 Frank Broidoux,2,3 Centre national de référence amylose AL et autres maladies par dépôts d’lg monoclonales 1Centre Hospitalier Universitaire de Poitiers, Poitiers, France; 2Centre National de la Recherche Scientifique, Limoges, France; 3Centre Hospitalier Universitaire de Limoges, Limoges, France.

Background: The pathophysiological mechanisms of proliferative glomerulonephritis with monoclonal immunoglobulin deposits (PGNMID) are still largely unknown. Only 30% of PGNMID cases have a detectable circulating monoclonal immunoglobulin (Ig) and a bone marrow corresponding clone.

Methods: We reviewed a French cohort of PGNMID with particular focus on hematological characteristics. A high-throughput sequencing assay from bone marrow and/or blood mRNA encoding immunoglobulins (RACE-RepSeq) was used to detect the underlying clone.

Results: Seventy-one patients (M/F ratio=1:6, median age 59 years) were included. At diagnosis, 73% had renal insufficiency (median serum creatinine=1.7 mg/dL). All patients had proteinuria, with nephrotic syndrome in 59% and microscopic hematuria in 85% of cases. No patient had extra-renal manifestations. By light microscopy, kidney biopsy revealed membranoproliferative glomerulonephritis (74%), mesangial glomerulonephritis (14%) or membranous glomerulonephritis (12%). By immunofluorescence (IF), deposits stained for IgG in 55 cases (mostly IgG3), IgM in 7 cases, IgA in 4 cases or light chain (LC) only in 5 cases. Serum and/or urine immunofixation was positive in 26 cases (37%). An underlying clone was found in 21 cases (30%) using bone marrow or blood flow cytometry analysis. The clonal detection rate was particularly low in IgG3-PGNMID (9%). The nature of the clone differed with PGNMID subtype: lymphoplasmacytic in IgM-PGNMID, and plasmacytic in IgA-LC-PGNMID. RACE-RepSeq analysis failed to detect a bone marrow or blood clone in 18/26 cases (IgG3-PGNMID, n=17; IgGAK-PGNMID, n=1). IF analysis of kidney samples using anti-Vκ antibodies showed positive staining for Vκ in 43/47 tested IgG3k-PGNMID patients without a detectable clone, whereas deposits stained only for Vk2 in one IgG1k-PGNMID patient who had a bone marrow Vk2 clone by RACE-RepSeq analysis.

Conclusions: These results suggest that PGNMID is a heterogeneous medical condition and that some cases might involve oligoclonal production of nephrotic Ig restricted to the IgG3k isotype. Such cases should no longer be classified as MGRS.

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Rituximab-Associated Flare of Cryoglobulinemic Vasculitis

Janina Paula T. Sy-Gio,1 Charat Thongsprayoon,2 Ziad Zoghby,3 Nelson Leung, Sandhya Manohar. Mayo Clinic Minnesota, Rochester, MN.

Background: Patients with cryoglobulinemic vasculitis (CV) can develop disease flare after rituximab administration. The pathogenesis is hypothesized to be from immune complex deposition in the microvasculature, wherein the immune complex consists of the involved cryoglobulin and an antigenic portion of rituximab. Our objective was to describe the prevalence, clinical characteristics, predisposing factors, and outcomes of rituximab-associated flare of CV.

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