

Department of Internal Medicine 34th Annual Yash P. Kataria Research Day

Sample Abstract Form

DEADLINE: Tuesday, January 28, 2020

Email abstract to Cindy Kukoly, kukolyc@ecu.edu

PLEASE NOTE

Your abstract must fit in the text box to the right using Arial font, 9 point type. Please see the submission guidelines for more information on properly submitting your abstract.

You may not change the size of the text box, alter the format, adjust the font, type size, etc.

<u>Reminder-</u> For trainees, please include mentor information below. Also be sure to cc mentor in email at time of abstract submission.

Failure to follow these instructions may delay acceptance or disqualify your abstract.

Notification of Acceptance

February 4th

Formatting questions? Call Cindy Kukoly at 252-744-2962 or Cathy Munson at 252-744-5258

RITUXIMAB DEPLETES	B-LYMPHOCYTES IN PULMONARY	ALVEOLAR
PROTEINOSIS		<u>H</u>

H Dalrymple, A Malur, G Gagnon, I Marshall, S Arce, BP Barna, MS Kavuru, MJ Thomassen

Background: Pulmonary Alveolar Proteinosis (PAP) is an autoimmune disorder characterized by autoantibodies to GM-CSF. Rituximab, a chimeric murine-human monoclonal antibody, directed against the B lymphocyte specific antigen CD20, has shown promise in a number of autoimmune disorders. We hypothesized that Rituximab would deplete B-lymphocytes in PAP and thus improve clinical condition.

Methods: As part of an open label proof-of-concept Phase II clinical trial of 10 patients, to date, 7 PAP patients received two infusions (1mg/ml) of Rituximab, fifteen days apart. Peripheral blood samples were collected from patients both pre- and post-Rituximab infusion. Lymphocyte activation was determined by measurement of ATP uting CD4 migrations by ad a says (Cylex). Peripheral blood mononucle in cells (PBMC) were also is plate from both PAP patients and healthy controls and an alysed by yow of tome try. Oxygenation of PAP patients was measured by PaO₂.

Results: B-lymphocytes decreased by 15±2 % (n=7) by fifteen days post therapy and persisted for 6 months. T-lymphocyte activity increased by 55% following administration of Rituximab, persisting three months post therapy (n=5), as compared to <1% increase in activity of T-lymphocytes from healthy controls (n=4). Oxygenation was also determined to be improved by 14±5 mmHg in 4/5 patients.

Conclusions: Administration of Rituximab to PAP patients effectively depleted B- lymphocytes and enhanced T-lymphocyte activity, suggesting that in PAP, Rituximab affects both humoral and cellular immune systems. Rituximab also improved clinical symptoms, indicating that therapy directed at the autoimmune pathogenesis of PAP represents an effective and beneficial approach.

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