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N.M. Kuderer: Advisory/Consultancy, Travel/Accommodation/Expenses: BMS; Advisory/Consultancy, Travel/Accommodation/Expenses: Janssen; Honoraria (self), Advisory/Consultancy: Celldex; Honoraria (self), Advisory/Consultancy: Invitae; Honoraria (self), Advisory/Consultancy: Total Health; Honoraria (self), Advisory/Consultancy: Beyond Springs; Honoraria (self), Advisory/Consultancy: BAyer; Honoraria (self), Advisory/Consultancy: Spectrum Pharmaceuticals. S. Gulati: Advisory/Consultancy: Puma Biotechnology; Travel/Accommodation/Expenses: ASCO; Research grant/Funding (self): AstraZeneca; Research grant/Funding (self): IsoRay. T.K. Choueiri: Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): AstraZeneca; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Alexion; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Bayer; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): BMS; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Cerulean; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Eisai; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Foundation Medicine; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Exelixis; Advisory/Consultancy, Research grant/Funding (self): Ipsen; Research grant/Funding (self): Tracon; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Genentech; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Roche; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): GSK; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Lilly; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Merck; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Novartis; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Peloton; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Pfizer; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Promethius Labs; Advisory/Consultancy, Research grant/Funding (self): Corvus; Research grant/Funding (self): Calithera; Honoraria (self), Advisory/Consultancy, Research grant/Funding (self): Analysis Group; Honoraria (self), Research grant/Funding (self): Sanofi/Aventis; Research grant/Funding (self): Takeda; Honoraria (self): EMD Serono; Honoraria (self): Navinata Healthcare; Honoraria (self), Advisory/Consultancy: NCCN; Honoraria (self): Michael J. 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LBA72 Assessment of clinical and laboratory prognostic factors in patients with cancer and SARS-CoV-2 infection: The COVID-19 and Cancer Consortium (CCC19)

P. Grivas[1], J. Warner[2], Y. Shyr[3], D. Shah[4], S.M. Rubinstein[5], N.M. Kuderer[6], T.K. Choueiri[7], D. Rivera[8], C. Painter[9], M.A. Thompson[10], S. Peters[11], A. Desai[12], A.R. Khaki[13], N.A. Pennell[14], J. Hawley[15], B. Halmos[16], M.M. Puc[17], G.H. Lyman[18], B.I. Rini[19], G. Lopes[20]

[1]Medicine, Division of Oncology, University of Washington, Fred Hutchinson CRC, Seattle Cancer Care Alliance, Seattle, WA, USA; [2]Medicine, Institution/Organization Vanderbilt University Medical Center, Nashville, TN, USA; [3]Biostatistics, Institution/Organization Vanderbilt University Medical Center, Nashville, TN, USA; [4]Population Health Sciences, Mays Cancer Center at UT Health San Antonio MD Anderson, San Antonio, CA, USA; [5]Medicine, Division of Hematology, Vanderbilt University Medical Center, Nashville, TN, USA; [6]Medical Oncology, Advanced Cancer Research Group, Seattle, WA, USA; [7]Medical Oncology Dept., Dana Farber Cancer Institute, Boston, MA, USA; [8]Division of Cancer Control and Population Sciences, National Cancer Institute, Rockville, MD, USA; [9]Cancer Program, Broad Institute, Cambridge, MA, USA; [10]Aurora Cancer Care, Advocate Aurora Health, Milwaukee, CO, USA; [11]Multidisciplinary Oncology Center, Centre Hospitalier Universitaire Vaudois - CHUV, Lausanne, Switzerland; [12]Medicine, University of Connecticut, Farmington, CT, USA; [13]Medicine, University of Washington, Seattle, WA, USA; [14]Hematology and Medical Oncology, Cleveland Clinic, Cleveland, OH, USA; [15]Medicine, Columbia University Irving Medical Center, New York, NY, USA; [16]Department of Oncology, Montefiore/Albert Einstein Cancer Center, Bronx, NY, USA; [17]Surgery, Virtua Health, Marlton, NJ, USA; [18]Public Health Sciences, Fred Hutchinson Cancer Research Center, Seattle, WA, USA; [19]Division of Hematology/Oncology, Vanderbilt University Medical Center, Nashville, TN, USA; [20]Sylvester Comprehensive Cancer Center, University of miami, Miami, FL, USA

Background: The impact of clinicopathologic factors, cancer type, stage or therapies on outcomes of pts with COVID19 is not well defined. We systematically and comprehensively identified and assessed factors associated with high mortality (M) in the largest cohort of pts with cancer and COVID-19.

Methods: CCC19 cohort includes pts with active or prior cancer and COVID-19 across US/international sites and collaborates with ESMO-CoCARE. Analysis was limited to lab-confirmed COVID-19. Primary endpoint: all-cause 30-day M. Multivariable logistic regression was used to assess association between 30-day M and *a priori* identified demographic/clinicopathologic risk factors (age, sex, race, region, smoking, obesity, comorbidities, ECOG PS, cancer status, recent [in 3 months] cancer treatment, cancer type, baseline COVID19 severity). Exploratory analysis used separate models adjusted for demographic/clinicopathologic factors to assess associations of lab parameters with 30-day M.

Results: As of 31 July 2020, 4169 pts have been accrued; median follow-up 30 days (IQR 21-70), median age 66 (IQR 56-76), 50% men, 92% from US, breast and prostate cancer were most common; 38% had active cancer, 56% required hospitalization and 16% ICU. In 3830 pts with lab confirmed COVID19, 30-day M was 14% overall and 23% in hospitalized pts. Table shows adjusted [a]OR for overall and hospitalized pts. Age, male sex, smoking, >2 comorbidities, ECOG PS≥1, progressive cancer, hematologic or >1 cancer, and severe baseline COVID19 at presentation were associated with worse 30-day M. In hospitalized pts, high or low ALC, high ANC, low platelets, abnormal creatinine, d-dimer, HS-troponin and CRP were also associated with worse 30-day M.

Table: LBA72

	OVERall (N = 3819)	Hospitalized (N = 2168)
Age	1.6 (1.4-1.6)	1.6 (1.4-1.6)
Male	1.3 (1.0-1.6)	1.3 (1.0-1.6)
Ever Smoker	1.3 (1.0-1.6)	0.8 (0.6-1.0)
>2 Comorbidities	2.0 (1.1-3.6)	1.9 (1.0-3.5)
ECOG PS 1	1.8 (1.3-2.6)	0.6 (0.4-0.8)
ECOG PS >1	3.5 (2.5-5.0)	1.8 (1.3-2.4)
progressIVE CA	2.6 (1.8-3.7)	2.4 (1.7-3.5)
Recent Therapy	1.4 (1.0-1.8)	1.4 (1.0-1.8)
HemE CA	1.4 (1.0-1.8)	1.2 (0.9-1.6)
>1 ca	1.4 (1.0-1.9)	1.2 (0.9-1.7)
Mod C19	5.5 (3.9-7.7)	0.7 (0.4-1.0)
Sev C19	23.4 (16.1-34.1)	4.1 (3.1-5.3)
LABs		
ALC>ULN		2.1 (1.0-4.2)
ALC<LLN		1.4 (1.1-1.9)
ANC>ULN		1.9 (1.4-2.5)
PLT<LLN		1.4 (1.1-1.8)
AB CREATInine		1.5 (1.2-2.0)
AB D-DIMER		2.0 (1.2-3.5)
AB HS-TROP		2.1 (1.3-3.5)
AB CRP		2.1 (1.1-4.2)

*AB=abnormal.

Conclusions: We confirmed *a priori* identified risk factors for poor prognosis in the largest COVID-19/cancer cohort and performed initial analysis of lab parameters, informing risk assessment.

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Legal entity responsible for the study: The COVID-19 and Cancer Consortium (CCC19).

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J. Warner: Advisory/Consultancy: IBM Watson Health; Advisory/Consultancy: Westat; Leadership role, Shareholder/Stockholder/Stock options: HemOnc.org LLC; Travel/Accommodation/Expenses: American Society of Clinical Oncology; Research grant/Funding (institution): National Cancer Institute. Y. Shyr: Advisory/Consultancy: Roche; Advisory/Consultancy: Novartis; Advisory/Consultancy: Pfizer; Advisory/Consultancy: Johnson & Johnson; Advisory/Consultancy: GlaxoSmithKline; Advisory/Consultancy, Speaker Bureau/Expert testimony: AstraZeneca; Speaker Bureau/Expert testimony: Boehringer Ingelheim; Speaker Bureau/Expert testimony: Eisai. D. 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A.R. Khaki: Shareholder/Stockholder/Stock options: Pfizer; Shareholder/Stockholder/Stock options: Merck. N.A. Pennell: Advisory/Consultancy: Astrazeneca; Advisory/Consultancy: Merck; Advisory/Consultancy: Eli Lilly; Advisory/Consultancy: Genentech; Advisory/Consultancy: BMS; Advisory/Consultancy: Amgen. B. Halmos: Advisory/Consultancy, Research grant/Funding (institution): AstraZeneca; Advisory/Consultancy, Research grant/Funding (institution): Bristol Myers Squibb; Advisory/Consultancy, Research grant/Funding (institution): Merck; Advisory/Consultancy, Research grant/Funding (institution): Boehringer-Ingelheim; Advisory/Consultancy, Research grant/Funding (institution): Novartis; Advisory/Consultancy: Pfizer; Advisory/Consultancy, Research grant/Funding (institution): Amgen; Advisory/Consultancy: Spectrum; Advisory/Consultancy: Takeda; Advisory/Consultancy, Research grant/Funding (institution): Guardant Health; Advisory/Consultancy: Foundation One; Advisory/Consultancy, Research grant/Funding (institution): Genentech; Research grant/Funding (institution): AbbVie; Research grant/Funding (institution): Advaxis; Research grant/Funding (institution): GSK; Research grant/Funding (institution): Eli-Lilly; Research grant/Funding (institution): Mirati. G.H. 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Rini: Advisory/Consultancy, Research grant/Funding (institution), Travel/Accommodation/Expenses: Merck; Advisory/Consultancy, Research grant/Funding (institution): Roche; Advisory/Consultancy, Research grant/Funding (institution), Travel/Accommodation/Expenses: Pfizer; Advisory/Consultancy, Research grant/Funding (institution): AVEO; Advisory/Consultancy: Arravive; Advisory/Consultancy, Research grant/Funding (institution), Travel/Accommodation/Expenses: BMS; Advisory/Consultancy: 3D Medicines; Advisory/Consultancy: Synthorx; Advisory/Consultancy: Surface Oncology; Shareholder/Stockholder/Stock options: PTC Therapeutics; Research grant/Funding (institution): AstraZeneca. G. Lopes: Honoraria (self), Travel/Accommodation/Expenses: Boehringer Ingelheim; Advisory/Consultancy, Research grant/Funding (institution), Travel/Accommodation/Expenses: Pfizer; Advisory/Consultancy, Research grant/Funding (self), Research grant/Funding (institution): AstraZeneca; Research grant/Funding (institution): Merck Sharp & Dohme; Research grant/Funding (institution): EMD Serono; Research grant/Funding (institution): Blueprint Medicines; Research grant/Funding (institution): Tesaro; Research grant/Funding (institution): Bavarian Nordic; Research grant/Funding (institution): Novartis; Research grant/Funding (institution): G1 Therapeutics; Research grant/Funding (institution): Adaptimmune; Research grant/Funding (institution): BMS; Research grant/Funding (institution): GSK; Research grant/Funding (institution): AbbVie; Research grant/Funding (institution): Rgenix; Research grant/Funding (institution): Roche; Research grant/Funding (institution): Genentech; Research grant/Funding (institution): Lilly; Research grant/Funding (institution), Travel/Accommodation/Expenses: Janssen; Travel/Accommodation/Expenses: E.R. 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LBA73 The ORF1ab of SARS-CoV-2 encodes an immunodominant epitope restricted by HLA-A*01:01

P. Kvistborg[1], A. Gangaev[1], S. Ketelaars[1], S. Patiweal[1], A. Dopler[1], O. Isaeva[1], K. Hoefakker[1], S. De Biasi[2], C. Mussini[2], G. Guaraldi[2], M. Girardis[2], C. Talavera Ormeno[3], P. Hekking[3], N. Lardy[4], M. Toebes[1], R. Balderas[5], T.N. Schumacher[6], H. Ovaa[3], A. Cossarizza[2]

[1]Immunology, NKI-AVL - Netherlands Cancer Institute/Antoni van Leeuwenhoek Hospital, Amsterdam, Netherlands; [2]Department of Medical and Surgical Sciences for Children and Adults, University of Modena, Modena, Italy; [3]Department of Cell and Chemical Biology, Leiden University Medical Center, Leiden, Netherlands; [4]Department of Immunogenetics, Sanquin Diagnostics, Amsterdam, Netherlands; [5]Department of Biological Sciences, BD Bioscience, San Jose, CA, USA; [6]Immunology, Netherlands Cancer Institute/Antoni van Leeuwenhoek Hospital (NKI-AVL), Amsterdam, Netherlands

Background: A large global effort is ongoing to develop vaccines against SARS-CoV-2, the causative agent of COVID-19. While there is accumulating information on the antibody response against SARS-CoV-2, less is known about the SARS-CoV-2 antigens that are targeted by CD8 T cells. Such knowledge will be of high value to gain fundamental insights into the antigenic landscape of SARS-CoV-2 recognized by CD8 T cells, to develop tool allowing focused analysis of the SARS-CoV-2 specific T cell responses directly ex vivo, and to understand whether current vaccine designs are covering the CD8 T cell recognized antigens.

Methods: To address this issue, we have analyzed samples from 18 COVID-19 patients for CD8 T cell recognition of 500 predicted SARS-CoV-2-derived epitopes restricted to 10 common HLA-A and HLA-B alleles. For each HLA allele, the top 50 epitopes were selected based on predicted binding affinity and likelihood of successful proteasomal processing. To probe for CD8 T cell recognition of the selected epitope-HLA complexes, we made use of our in-house technology based on multiplexing of peptide HLA (pHLA) multimers conjugated to fluorescent dyes.

Results: In addition to previous studies showing CD8 T cell reactivity towards epitopes derived from the spike protein of SARS-CoV-2, we have identified several CD8 T cell recognized epitopes derived from the ORF1ab, including one epitope displaying clear immunodominant properties across patients positive for HLA-A*01:01. Investigation of the functional status of part of the identified responses (including 4 responses specific for the immunodominant epitope) revealed that the T cell responses were