deconditioning; 90% (37/41) were discharged within one appointment. One-third (13/41) were later either referred back to secondary care (8/41), frequented acute medical services (3/41) or accessed further specialist testing through secondary care (9/41) for the same complaint of breathlessness. Only one re-referral led to a new diagnosis (of COPD, six years after CPET) and of 29 additional investigations for the nine patients who underwent repeat testing, no additional diagnoses were found.

4% (2/41) were later diagnosed with a pathology potentially 'missed' by their CPET. One patient had a paroxysmal arrhythmia, not present at time of CPET. The second presented four months after CPET with myocardial infarction; their dysfunctional breathing pattern was so pronounced at CPET that they couldn't reach adequate levels of exercise to reveal ischaemia.

Conclusions This study provides support for use of CPET to reliably exclude harmful pathology and reassure both patients and clinicians early within a secondary care pathway for unexplained breathlessness. The diagnostic limitations of a submaximal test must be appreciated.

COVID-19: contact, admission, recruitment and outcome

P251 THE INDUCTION OF EARLY, DYNAMIC AIRWAY MUCOSAL AND SYSTEMIC IMMUNE RESPONSES FOLLOWING RECENT SARS-COV-2 HOUSEHOLD EXPOSURE

TD Pillay, A Kondratiuk, M Davies, JS Narean, C Tejpal, L Wang, L Kavege, C Memmi, S George, J Zhou, C Rosadas, R Varro, A Rowan, C Herrera, G Taylor, M McClure, W Barclay, J Fenn, R Kundu, S de Lusignan, A Lalvani, On behalf of the Integrated Network for Surveillance, Trials and Investigation of COVID-19 Transmission (INSTINCT) study team. *Imperial College London, London, UK*

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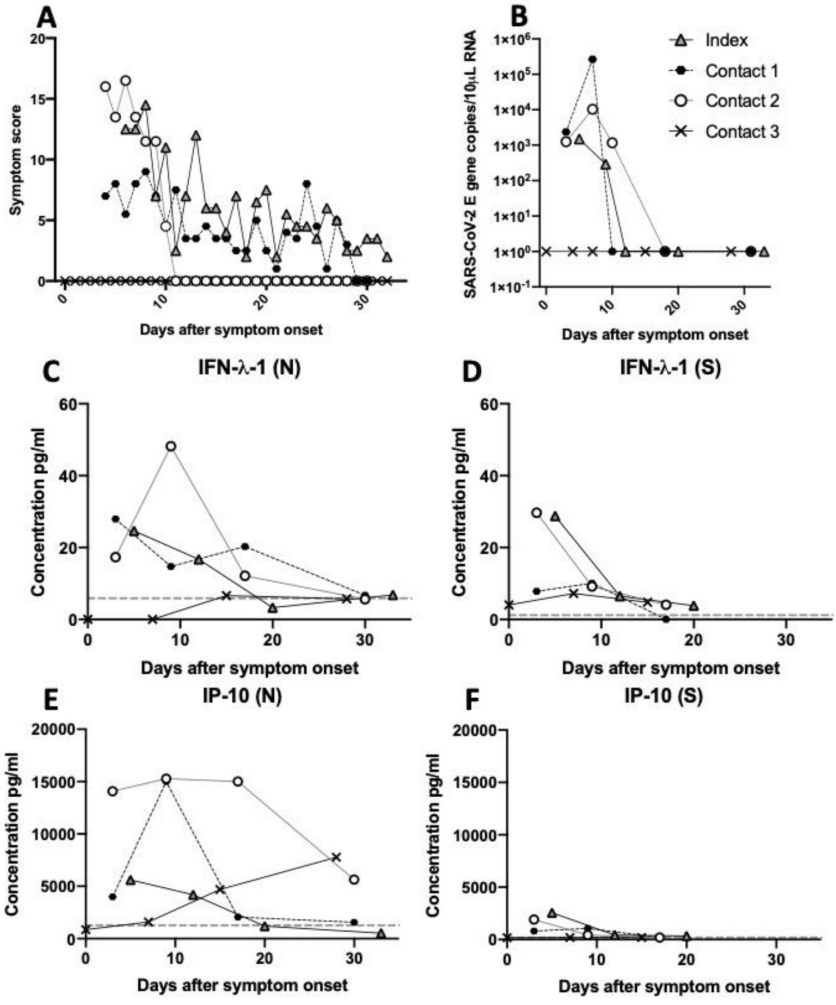
Objectives The wide spectrum of clinical outcomes to SARS-CoV-2 exposure suggests that early immune responses play a pivotal role.[1] We aim to describe early, longitudinal, local (nasal mucosal lining fluid) and systemic (peripheral blood) cytokine and cellular immune responses to SARS-CoV-2 in a



Abstract P251 Figure 1 Symptom score, virology, serology and nasal & serum cytokine data in the index case and their two PCR-positive household contacts since day of symptom onset at 4 timepoints across 28 days of follow up. **A.** Symptom score was calculated by allocating values for each self-reported symptom, weighted by self-reported severity, from a daily tracker; **B.** virology was measured by oropharyngeal swab RT-PCR. Samples below the detectable level were assigned value of 1; **C-F**. concentrations of 2 of the cytokines (IFNA1 and IP-10) measured in nasal lining fluid (**C & E**) and serum (**D & F**), measured by Meso Scale Discoveries U-plex assay. Grey dashed line indicates mean of healthy control values (**C&E**: n=4; **D&F**: n=5). Serum values for the fourth timepoint were not reported due to delayed sample processing.

symptomatic index case and their household contacts with detailed clinical and virological phenotyping. We hypothesise that immune responses at symptom onset would correlate with outcomes.

Methods Participants from the London area are referred to INSTINCT study by general practitioners as suspected, or Public Health England as laboratory-confirmed, cases (ethical review details: IRAS 282820, approved 24.04.2020). Households are visited the day after identification and again on days 7, 15 and 28. Clinical and exposure questionnaires, samples of environment (surface swabs and air); oropharynx (swabs); nasal mucosa (synthetic absorptive matrix) and blood, and daily symptom diaries are collected. Samples are analysed by PCR, serology, 20-plex cytokine assay and flow cytometry in institutional laboratories.

Results The index case was the first SARS-CoV-2 PCR-positive recruit of INSTINCT, confirmed on oropharyngeal swab 5 days after symptom onset. Contacts 1 and 2, the spouse and daughter, became symptomatic 2 days after the index case and were confirmed PCR-positive 3 days after symptom onset. The three PCR-positive individuals seroconverted during follow-up. Contact 3, the son, remained asymptomatic, PCR- and serology-negative throughout (figure 1a-b). None required hospitalisation. Swabs of the kettle and fridge handles were positive for virus, while other household surfaces and air samples were negative. Induction, peak and decline of interferon-λ-1 and IP-10 levels were captured in nasal mucosa, with lower serum levels (figures 1c-f).

Conclusion These data demonstrate the ability of the INSTINCT household contact study to capture early immune responses in mild SARS-CoV-2 infection, not captured by COVID-19 hospital cohort studies. Early nasal mucosal cytokine responses to SARS-CoV-2 infection are not reflected in serum. The correlations observed provide cogent hypotheses that will be tested in the larger INSTINCT cohort, with implications for COVID-19 risk stratification, therapeutics, prophylaxis and vaccinology.

REFERENCE

1. Vabret N, Britton GJ, Gruber C, *et al*. Immunology of COVID-19: Current State of the Science. *Immunity*. 2020;**52**(6):910–41.

P252 ROLE OF CHEST X-RAY IN DIAGNOSIS AND PREDICTING OUTCOME OF COVID19 INFECTION IN A DISTRICT GENERAL HOSPITAL SETTING

A Sivaramakrishnan, M Syed, E Phyu, J Kuzhively, D Betarse, V Patil. *Milton Keynes University Hospital NHS Trust, Milton Keynes, UK*

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Introduction We set out to look at the role of chest x-ray (CXR) in diagnosing novel coronavirus (Covid19) infection and to explore if it can predict clinical outcomes. We compared the chest imaging and swab results in Covid19 patients. Demographics, symptoms and CXR findings were explored as predictors of clinical outcome in COVID19 patients.

Methods All adult patients who had CXR and Reverse Transcriptase Polymerase Chain Reaction (RT-PCR) available between March and June 2020 were included. Data was collected retrospectively from electronic case notes. CXR reported as typical or atypical COVID features[1] was considered as positive. For predictors of outcomes, regression analysis was conducted.

Results 876 patients had both CXR and RT-PCR swabs. Their mean age was 64.6 years and age range was 17 to 105 years. 324 (37%) were positive and 552 (63%) negative on RT-PCR. CXR showed typical COVID19 changes in 217 (24.8%) and atypical COVID19 changes in 148 (16.9%) patients. The sensitivity and specificity of CXR in the overall study group was 59.9% and 69% with positive predictive value (PPV) and negative predictive value (NPV) of 53.2% and 74.6% respectively. 692 patients were admitted and the sensitivity and specificity of CXR in this group was 66.9% and 65.5% with PPV and NPV of 53% and 77.4% respectively. 148 patients (16.8%) received ventilator support (27 received invasive and 121 had non-invasive ventilator support) and the sensitivity and specificity of CXR in this sub-group was 78% and 42.4% with PPV and NPV of 62.7% and 60.9%. There were 129 deaths and on multivariate regression analysis, age (> 60 years) and positive CXR remained significant risk factors for the clinical outcome (Table). In contrast to the current literature, our sample did not show gender as a risk factor to predict the outcome.

Conclusions CXR has a reasonable specificity and sensitivity for diagnosing COVID19 and it increases with the severity especially in patients needing ventilator support. CXR can be used in predicting worse clinical outcome in COVID19 pneumonia.

REFERENCE

1. BSTI COVID-19 guidance for the reporting radiologists, https://www.bsti.org.uk/covid-19-resources/

P253 ACUTE PULMONARY EMBOLI AND COVID-19

N Johl, D Ap-Emyr, M Jones, K Hardingham, E King, C Carder, A Marin, C Corscadden, K Pink, J Underwood. *Cardiff and Vale University Health Board, Cardiff, UK*

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Abstract P252 Table 1 Regression analysis of age, gender, presenting symptoms and CXR for clinical outcomes (death or discharged)

			Univariate			Multivariate		
	Death	Discharge	Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
Age (>60 years)	113/129 (87.6%)	409/736 (55.6%)	5.145	3.097 – 8.547	<0.001	5.150	3.024 – 8.772	<0.001
Sex (Male)	72/129 (55.8%)	202/736 (27.4%)	1.314	0.903– 1.911	0.153	1.212	0.812– 1.810	0.346
Fever	52/129 (40.3%)	139/736 (18.9%)	0.842	0.576 – 1.229	0.373	0.900	0.584 – 1.385	0.631
SoB	78/129 (60.5%)	195/736 (26.5%)	1.363	0.931 – 1.997	0.112	1.235	0.803 – 1.899	0.337
Cough	52/129 (40.3%)	146/736 (19.8%)	0.773	0.529 – 1.131	0.186	0.816	0.527 – 1.264	0.363
CXR	87/129 (67.4%)	146/736 (19.8%)	3.396	2.289 – 5.039	<0.001	3.455	2.245 – 5.316	<0.001