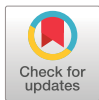SHAREABLE PDF

Intrapulmonary shunt and alveolar dead space in a cohort of patients with acute COVID-19 pneumonitis and early recovery

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Abstract

Background Pathological evidence suggests that coronavirus disease 2019 (COVID-19) pulmonary infection involves both alveolar damage (causing shunt) and diffuse microvascular thrombus formation (causing alveolar dead space). We propose that measuring respiratory gas exchange enables detection and quantification of these abnormalities. We aimed to measure shunt and alveolar dead space in moderate COVID-19 during acute illness and recovery.

Methods We studied 30 patients (22 males; mean±SD age 49.9±13.5 years) 3–15 days from symptom onset and again during recovery, 55±10 days later (n=17). Arterial blood (breathing ambient air) was collected while exhaled oxygen and carbon dioxide concentrations were measured, yielding alveolar–arterial differences for each gas (P_{A-aO_2} and P_{a-ACO_2}, respectively) from which shunt and alveolar dead space were computed.

Results For acute COVID-19 patients, group mean (range) for P_{A-aO_2} was 41.4 (−3.5–69.3) mmHg and for P_{a-ACO_2} was 6.0 (−2.3–13.4) mmHg. Both shunt (% cardiac output) at 10.4% (0–22.0%) and alveolar dead space (% tidal volume) at 14.9% (0–32.3%) were elevated (normal: <5% and <10%, respectively), but not correlated (p=0.27). At recovery, shunt was 2.4% (0–6.1%) and alveolar dead space was 8.5% (0–22.4%) (both p<0.05 *versus* acute). Shunt was marginally elevated for two patients; however, five patients (30%) had elevated alveolar dead space.

Conclusions We speculate impaired pulmonary gas exchange in early COVID-19 pneumonitis arises from two concurrent, independent and variable processes (alveolar filling and pulmonary vascular obstruction). For most patients these resolve within weeks; however, high alveolar dead space in ~30% of recovered patients suggests persistent pulmonary vascular pathology.