LETTER

Silent hypoxia is not a distinguishing feature of COVID-19 patients.

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ABSTRACT

Patients with and without COVID-19 have a wide range of physiological responses to hypoxaemia. COVID-19 patients are not more physiologically "happy" in response to hypoxia, and have greater respiratory rates for any given SpO2/FiO2 ratio. In COVID-19, decreasing physiological "happiness" in response to hypoxaemia is related with worse results. From bedside observations, the ROX index may be a straightforward tool for distinguishing patients at risk of bad outcomes in COVID-19.

Key Words: *Hypoxaemia.*

INTRODUCTION

To describe individual patient's physiological response to COVID-19 pneumonitis, the syndrome of "silent hypoxaemia", hypoxaemia that is well tolerated with less dyspnea than the treating clinician expects and the corresponding colloquialism "happy hypoxia" have been introduced in both clinical and journalistic settings. However, it is uncertain if silent hypoxaemia is a real condition or only a moniker applied to individual individuals who are notable outliers in terms of their predicted respiratory response to hypoxaemia, regardless of SARS-CoV-2 infection status. Although the term "asymptomatic hypoxaemia" was coined to describe patients with COVID-19 who did not have dyspnea in the context of severe hypoxaemia, objective measurements of respiratory drive (tidal volume or mean inspiratory flow) or subjective assessments of work of breathing are frequently under documented in clinical practice for patients outside of the ICU. As a result, the physiological reaction to hypoxaemia, as recorded by nursing staff as regular observations, is increasingly being seen as a proxy for "happy" or, in the case of SARS-CoV-2 infection, as a signal for a lack of abnormalities in general breathing pattern. Although initial recommendations cautioned against treating hypoxemia without evidence of respiratory distress, subsequent studies found that silent hypoxaemia was a risk factor for poor outcomes. Hypoxaemia relative to percentage of inspired oxygen (FiO2) is regarded to be a stronger predictor of COVID-19 severity than absolute hypoxaemia, as measured by the SpO2/FiO2. Ratio of SFR. The SFR is used similarly to the PaO2/FiO2 ratio in ventilated patients to quantify the severity of Acute Respiratory Distress Syndrome (ARDS), with lower values representing a deteriorating degree of hypoxaemia relative to inspired oxygen. The Respiratory Rate Oxygenation (ROX) index, or the ratio of SFR to respiratory rate which was developed to measure the probability of high-flow oxygenation failure in all-comers with respiratory failure, may be predictive of the requirement for intubation in COVID-19 patients. The relationship between a greater respiratory rate for a given degree of relative hypoxaemia (and hence a lower ROX score) and non-invasive oxygenation failure contradicts the worry that asymptomatic hypoxaemia may be linked with negative consequences. As such, we sought to determine whether a distinct physiological phenotype of "happy hypoxia" in COVID-19 patients was an identifiable clinical entity by comparing the physiological response to absolute hypoxaemia (measured by peripheral oxygen saturation, SpO2, regardless of inspired oxygen) and relative hypoxaemia (by calculating the SFR) between COVID-19 patients and those who had hypoxaemia respiratory failure from causes other than COVID-19. We also wanted to see if patients with COVID-19 who deteriorated (needed ICU within two weeks of diagnosis or died within 60 days) had a different physiological response to hypoxaemia, and if so, whether clinicians should be reassured, concerned, or unsure about a reduced physiological response to hypoxaemia. We conducted an observational cohort analysis of all adult patients admitted to a large acute hospital trust in the East Midlands, UK, with hypoxaemic respiratory failure. Patients with COVID-19 were compared to those who did not. A linear mixed effects model was used to simulate the physiological response to hypoxia. Our findings show that, while "asymptomatic" hypoxaemia can occur in any individual patient with respiratory failure, a physiological phenotype of "happy" is no more common in patients with SARS-CoV-2 infection than in hypoxaemic respiratory failure from other causes. Indeed, patients with hypoxaemic respiratory failure caused by COVID-19 have a higher respiratory rate on average than patients without COVID-19 infection for any given degree of absolute or

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relative hypoxaemia, albeit with significant variability in response within and between individual patients in both cohorts. Our findings disprove the concept that COVID-19 infected individuals are any more "happy" with hypoxaemia than non-COVID-19 patients, with a lower total ROX in COVID-19 patients suggesting a more significant physiological reaction in this group to hypoxaemia. We also discovered that within the COVID-19 cohort, individuals with a more physiologically "unhappy" phenotype, such as increased respiratory and heart rates at all levels of hypoxia, had poorer outcomes. This is corroborated by the fact that the ROX index is lower in individuals with poorer outcomes, indicating a link between a greater respiratory rate for any given degree of relative hypoxaemia and an increased risk of ICU admission or death. The idea that "happy hypoxia" is a low compliance subtype of the condition has been challenged. Although increasing estimated shunt fraction has been shown to be associated with mortality, this simply explains the degree of relative hypoxaemia observed in our patient group, with no evidence for any biochemical or ventilatory disease subtypes prior to the onset of mechanical ventilation in those requiring ICU. Furthermore, dyspnea is not always caused by acute hypoxaemia since respiratory centre activity is relatively modest in the absence of serious derangements of the respiratory mechanics, implying that ventilatory needs are satisfied. As a result, dyspnea – or even a higher respiratory rate – would suggest respiratory impairment as a result of the disease, rather than a result of the disease itself. Our study does have certain limitations. Although the case fatality rate was comparable between COVID-19 and non-COVID-19 patients, a higher proportion of COVID-19 patients required ICU admission, which may bias observations towards a "sicker" overall population of patients given that the COVID-19 cohort had a more pronounced relative hypoxaemia across all observations. The COVID-19 infected group was also younger and more likely to be of a non-white ethnicity, which might restrict generalizability. Individual patients with COVID-19 demonstrate a more symptomatic phenotype in response to hypoxaemia than those with other causes of hypoxaemic respiratory failure, but there is a wide range of responses. As a result, whereas silent hypoxaemia can occur in any patient suffering from hypoxaemic respiratory failure, it is not more common in individuals infected with SARS-CoV-2.