
COVID-19 with silent hypoxemia

SHORT CASE REPORT

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Some COVID-19 patients deteriorate rapidly and seemingly without warning. This is also true for relatively young patients who were previously healthy or who had only minor underlying conditions. We describe one such case, in which a man in his sixties developed rapidly progressive respiratory failure with severe hypoxia.

A man in his sixties developed a fever and cough and began to feel weak after returning from a skiing holiday in Central Europe. After nine days of illness, the man's family contacted his general practitioner and asked whether he should be

assessed by a doctor. On the telephone the relatives did not give the impression that the matter was urgent. They reported that the man was somewhat short of breath, but had eaten well at dinner and had later showered. He was smiling and watching TV, and was a little better than the day before. Nevertheless, the doctor felt that something was amiss and decided to visit the patient at home.

On examination, the patient was cyanotic but smiling bravely. He was calm, cooperated well and did not appear to be under particular strain. His breathing was almost silent. On auscultation, bilateral dry crackles were heard. Neither the patient's general condition nor his degree of respiratory distress were commensurate with his physiological measurements: his respiratory rate was 36 breaths/min (reference range 12–16 breaths/min) and oxygen saturation (SpO₂) 66 % (> 95 %). The patient had blood pressure of 120/80 mm Hg and pulse of 104 beats/minute in a sitting position, but the exertion of going out into the yard and getting into the ambulance left him without a palpable radial pulse. His respiratory rate increased to 48 breaths/min in the ambulance despite receiving 12 l/min O₂ via a mask. He was awake during the journey, but was intubated immediately upon arrival at hospital and was placed on a ventilator. X-rays of the lungs showed bilateral diffuse opacities. A test for SARS CoV-2 proved to be positive.

Discussion

The first sign of serious disease in patients with coronavirus infection is usually pneumonia with respiratory failure, and data from China show that the majority do not develop kidney, heart or liver failure until later in the disease course in intensive care (1). Patients develop what is referred to as 'silent hypoxemia' (2). Impaired pulmonary diffusion leads to a gradual fall in oxygen saturation. The cause of the respiratory failure is not clear.

Ground-glass opacities on CT suggest interstitial changes (3). Histopathological examination has revealed diffuse alveolar damage (4). A hypoxia-driven tachypnoea accompanied by relatively preserved lung elasticity gives rise to a high minute volume with pronounced hypocapnia. The pathophysiology is similar to that seen with hypobaric hypoxia at high altitude or in a low-pressure chamber (5). Hypoxia with accompanying hypocapnia generates no sensation of breathlessness – on the contrary, it may feel comfortable. Confusion often occurs, and the patient may lose situational awareness. Some patients have dyspnoea during speech. In our experience, breathing effort seems to be less severely affected than in patients with bacterial pneumonia or pulmonary oedema, and COVID-19 patients do not necessarily appear dyspnoeic until late in the disease course (2). Pathological lung sounds are not always heard on auscultation (2). Furthermore, most previously healthy individuals have well preserved cardiac function and maintain adequate blood pressure despite severe hypoxia (2). This is in contrast to patients with COPD exacerbation, sepsis with respiratory failure, decompensated heart failure or massive pulmonary embolism, in whom dyspnoea, laboured breathing, hypercapnia and hypotension are common.

Patients with COVID-19 often develop respiratory failure 8–14 days after symptom onset, with 'silent hypoxemia' and a high respiratory rate (1, 2). We have seen examples of patients going from being physiologically normal to decompensating just a few hours later. Suggestions from relatives that the situation is worsening should be taken extremely seriously. Increasing respiratory failure in cases of COVID-19 is difficult to assess by telephone or video call, and general practitioners and doctors in emergency medicine should have a low threshold for visiting patients at home. Physiological parameters must always be measured irrespective of general condition. An increased respiratory rate and fall in oxygen saturation are signs of increasingly impaired pulmonary diffusion. In patients with previously intact pulmonary function, a fall in oxygen saturation should always be considered serious, and the possibility of hospital admission should be discussed. The patient's breathing effort must be assessed in terms of frequency, depth, retractions and use of accessory muscles. A high minute volume is worrying, and is a sign that the patient is compensating for an increasing degree of diffusion failure. Admission of patients with unchanged physiological parameters may also be justified on the basis of the overall clinical picture. If the patient is weak or dehydrated, admission or prompt reassessment of the patient should be considered.

Patients showing changes in physiological parameters should be treated with care. These patients may show significant improvement in physiological parameters in association with oxygen therapy, but they remain critically ill. They should be moved with care, and must not walk or exert themselves in any other way. Patients should sit upright or lie semirecumbent to facilitate breathing (6). Oxygen therapy should be administered liberally, i.e. up to 12–15 l/min via a non-rebreather mask. In the event of an inadequate response to oxygen therapy, treatment should be attempted with high-flow continuous positive airway pressure (CPAP), or bag-valve-mask ventilation. The Air Ambulance department at Oslo University Hospital recommends attempting CPAP therapy during transport (6).

We were struck by just how easy it would have been to overlook the fact that this man was critically ill. We have also seen other patients progress from being in a clinically unremarkable state to decompensating over the course of a few hours. The patient described here had a high respiratory rate, but he was relaxed and there was a certain 'tranquility' about the whole situation. His relatives did not express any great anxiety and it would have been easy to get the impression that the patient was on the road to recovery. A home visit, clinical examination and pulse oximetry would prove to be crucial.

The patient's next of kin have consented to the publication of this article.

The article has been peer-reviewed.

LITERATURE

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