



Parathyroid glands and COVID-19; a neglected entity

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Implication for health policy/practice/research/medical education: The mechanisms interacted as the etiologies of hypocalcemia in SARS-CoV-2 are impaired intestinal absorption of calcium, disturbed secretion or impaired response to parathormone due to increased concentration of inflammatory cytokines, vitamin D insufficiency and hypoalbuminemia. Moreover, hypoxia-induce tissue injury with consequent rise in calcium influx are another possible mechanism. However, the effect of SARS-CoV-2 on parathormone secretion could explain the hypocalcemia of COVID-19 patients.

Keywords: Coronavirus disease, Parathyroid glands, COVID-19, SARS-CoV-2, cytokines

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Coronavirus disease 2019 (COVID-19) is an extremely contagious disease caused by Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), enveloped RNA virus depending to the family of Coronaviridae (1). Since, the global spread of COVID-19 was fast, directing to announce this disease as a pandemic in March 2020 by the World Health Organization (WHO). Sick people usually have mild disease in nearly eighty percent of cases. However, 15% of cases with this disease have moderate signs and symptoms, demanding hospital admission, while around five percent present the severe form of the disease, necessitating admission in the intensive care unit (ICU) (2). In spite of our knowledge about the clinical presentation, prognosis and patients' complications of this disease, the possible interaction between COVID-19 and the parathyroid glands is ill-understood. It is well-detected that SARS-CoV-2 produces a hypersensitive immune state and a subsequent extensive inflammation in several body organs, particularly by various cytokines in the lungs (3). It is possible that, cytokine-mediated inflammation disturbs the parathyroid glands, leading to impaired parathormone secretion. Additionally, SARS-CoV-2 has affected

The care of hyperparathyroidism or hypoparathyroidism conditions. Several investigations have shown some electrolyte abnormalities like hypocalcemia in SARS-CoV-2 patients (4,5).

During the COVID-19 era, various cases of serum calcium disturbance have been reported. For example, Puca et al reported a case of significant hypocalcemia

following SARS-CoV-2 in a female case, due to the significant vitamin D deficiency. They concluded a possible association among vitamin D deficiency and the intensity of SARS-CoV-2 (6).

In a recent study by Raesi et al, on 91 SARS-CoV-2 individuals versus 169 healthy cases, showed 59.3% of COVID-19 individuals had hypocalcemia at hospitalization since in control cases around 32.5% had low serum calcium concentration. They also showed that, the rate of mortality and intensive care unit hospitalization were considerably higher among the hypocalcemia group than cases of eucalcemia. This study could not show a noteworthy difference in the serum vitamin D and parathormone concentrations among the groups. Regarding the severity of the infection, they found 74.1% of cases in hypocalcemia group presented a serious disease, since 24.3% of cases in eucalcemic groups had severe infection. They finally concluded that, hypocalcemia should be accompanying by greater death possibility (7). Liu et al in a more recent study showed two third of their COVID-19 patients had hypocalcemia (8). This retrospective study consisted of 107 COVID-19 patients who divided into hypocalcemia and eucalcemic groups. Liu and colleagues showed 62.6% of cases had hypocalcemia. They showed in hypocalcemia group, the inflammatory biomarkers were lower. They particularly found serum calcium concentration had a negative association with leukocyte counts and some other biomarkers, while they showed a positive relationship with lymphocyte count and serum albumin. Importantly, cases with hypocalcemia had poor outcome. Accordingly,

they showed serum calcium could predict the prognosis of patients (8). The mechanisms interacted as the etiologies of hypocalcemia in SARS-CoV-2 are impaired intestinal absorption of calcium, disturbed secretion or impaired response to parathormone due to increased concentration of inflammatory cytokines, vitamin D insufficiency and hypoalbuminemia. Moreover, hypoxia-induce tissue injury with consequent rise in calcium influx are another possible mechanism (8,9). However, the effect of SARS-CoV-2 on parathormone secretion could explain the hypocalcemia of COVID-19 patients (9).

Author's contribution

HN is the single author of the manuscript.

Conflicts of interest

The author declares that he has no competing interest.

Ethical issues

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