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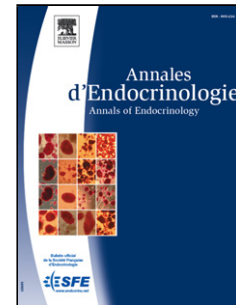
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Decompensated primary hypoparathyroidism in a patient with COVID-19

Un cas d'hypoparathyroïdie primitive décompensée chez un patient atteint de COVID-19

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Dear editor,

COVID-19 infection, through its emergence and contagiousness, is currently a major public health problem. It can be responsible of decompensation of pathologies that are in a balanced state outside of this aggression, e.g. phosphocalcic metabolism.

We report here the case of an 82 year old man hospitalized in a COVID-19 unit on November 10th, 2020. The patient had a corrected serum calcium level of 1.74mmol/l at the admission. Albuminemia (colorimetric technique) was 41g/l. Phosphoremia (0.94mmol/l) and magnesemia (0.82mmol/l) were within laboratory standards. Ionized calcium was low at 0.97mmol/l. Hypocalcemia was confirmed the next day at 1.68mmol/l with the rest of the calcium-phosphorus balance still within laboratory standards, 25-OH-Vitamin D was 111nmol/l. Vitamin D3, 1,25-Dihydroxy was 176.17pmol/ml (36.48-216.25). Serum parathormone was reduced to 0.954pmol/l (N 1.590 -6.893pmol/ml).

Initially the blood gases were in favor of respiratory alkalosis with pH at 7.49, O₂ and CO₂ blood pressure respectively at 64 mmHg and 29mmHg. CRP was at 123,60 mg/l with a moderate cytolysis (creatinine kinase: 390 IU/L, lipase: 97 IU/L and AST: 49 IU/L).

The patient was hospitalized 5 days from the onset of symptoms of SARS-COV-2 infection, with positive PCR. Chest CT scan reported lung involvement between 10 and 25% of the parenchyma, without pulmonary embolism.

Hypocalcemia was initially asymptomatic with a normal electrocardiogram (QT interval corrected to 420ms).

For COVID-19 disease, the patient was treated with DEXAMETHASONE 6mg for 12 days due to persistent oxygen reuptake. At 7 days from the onset of symptoms, an increase in oxygen up to 8L/min was needed with, nevertheless, excellent clinical tolerance. The patient was placed in prone position, leading to clinical improvement with a complete decrease in oxygen therapy in 4 days.

From the 4th day of hospitalization, despite oral supplementation, we noted a prolongation of the QTc segment at 470ms. Intravenous supplementation with 2g of Calcium hydrochloride/24h was therefore necessary for 4 days, leading to normalization of serum calcium and QTc. Intravenous supplementation was stopped with an oral relay by Calcium carbonate 4g/day and Alfacalcidol 1µg/day allowing normalization of calcemia for the duration of the hospitalization. Calcium level was measured on December 8th, 2020 at 2.90mmol/l, leading to stop the vitamin-calcium supplementation. On January 8th, 2021, the phosphocalcic balance was normal with a PTH adapted (2.12pmol/l).

No history of surgery, trauma or cervical radiation was found, neither polyendocrinopathy. The immunological assessment did not reveal any autoimmune field. The morphological assessment was normal as was the PET-scan.

Hypocalcemia has been described as common in COVID-19 disease (1) and seems to be a distinctive biochemical feature of COVID-19 compared to other acute respiratory distress syndromes (2). Moreover, It appears to be a predictive factor in the development of a severe form of COVID-19 (3). However, these findings appear to be primarily related to vitamin D deficiency. Indeed, there is a strong literature exploring the effect of vitamin D deficiency and supplementation on the occurrence and prognosis of viral respiratory infections (4). Although the impact of vitamin D supplementation on the prevalence of COVID-19 infection has not yet been demonstrated (5), supplementation appears to be associated with a better prognosis (6). The French-speaking society of clinical and metabolic nutrition (7) has recommended systematic vitamin D supplementation, even in the absence of deficiency, although the European Society of Nutrition (ESPEN) proposes it only for undernourished subjects (8).

These elements differ from the case presented here. The patient was not deficient in vitamin D. He did not suffer from severe chronic renal insufficiency. Inadequate PTH, therefore, acts as a primary hypoparathyroidism. We have a single serum calcium level of 2.05 mmol/l in December 2019 before the episode of SARS-Cov-2 infection. Albuminemia was not measured at that time to calculate corrected serum calcium, but we have no evidence of hypo-albuminemia. This hypocalcemia may be a marker of a well-tolerated old primary hypoparathyroidism.

Three previous similar cases of decompensation of frustrated primary hypoparathyroidism in patients with SARS-Cov-2 have been reported (9-11). Cases of glandular infiltration have been monitored in the early stages of the COVID-19 epidemic, particularly in tissues with ACE2 receptors (12, 13), which is not the case in the parathyroid gland (14). To our knowledge, we do not have any description of viral infiltration of SARS-Cov-2 in the parathyroid gland.

Conclusion

We report here the case of decompensation of a primary hypoparathyroidism during a COVID-19 disease. This observation should remind us that, although SARS-Cov-2 does not present a known tropism for the parathyroid gland, the severity of the infection can lead to decompensation of pathologies that were well tolerated before, even in the absence of vitamin D deficiency.

The patient has given his agreement for this clinical report.

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Availability of data and material: All data are available

Code availability: No software has been used

Authors' contributions: J-B. Bonnet and E. Berchoux wrote the first draft of the manuscript. Critical revision of the manuscript for important intellectual content: all co-authors. All authors have approved the final version of the manuscript.

Consent for publication: The patient has given his agreement for this clinical report.

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