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were within laboratory standards. Ionized calcium was low at 0.97 mmol/l. Hypocalcemia was confirmed the next day at 1.68 mmol/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 176.17 pmol/l (36.48-216.25). Serum parathormone was reduced to 0.954 pmol/l (N 1.590 - 6.893 pmol/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 29 mmHg. CRP was at 123.60 mg/l with a moderate cytolsis (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 3 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 with Calcium carbonate 4g/day and Alfacalcidol 1µg/day allowing normalization of calcium for the duration of the hospitalization.

Calcium level was measured on December 8th, 2020 at 2.90 mmol/l, leading to stop the vitamin-calcium supplementation. On January 8th, 2021, the phosphocalcic balance was normal with a PTH adapted (2.12 pmol/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 could be a marker of a well-tolerated old primary hypoparathyroidism.
Three previous similar cases of decompensation of 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.

1. Conclusion

We report here the case of decompensation of a mild form of 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.

Disclosure of interest

The authors declare that they have no competing interest.

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A new trigger in pheochromocytoma crisis: Giant leiomyoma

Le léiomyome géant : un nouveau facteur de crise de phéochromocytome

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1. Case report

A 57-year-old woman with personal background of uterine fibroids was referred for complaints of morning headache that required paracetamol treatment. Some hours later, she was attended by mobile unit emergency in response to coordination call centre 112. The patient suddenly suffered from palpitations, aphasia, dyspnea, pulmonary edema and psychomotor agitation. She presented hypoxia with oxygen saturation level (SpO2) below 80%. The events were triggered after an oral intake and a supine to prone position change. She required sedation to prevent agitation-related harm. On admission to the Intensive Care Unit, physical examination revealed a palpable mass in the upper right side of the abdomen. The blood pressure was 155/110 mm Hg. An electrocardiography showed sinus tachycardia (130 beats/min) and ST-segment elevation in inferior leads, the biochemical marker: