Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

[4] Visser WE, Friesema EC, Visser TJ. Minireview: thyroid hormone transporters: the knowns and the unknowns. Mol Endocrino 2011;25:1–14.
[5] Vanes NK, Lazarus JH, Chan S-Y. Thyroid function in pregnancy: maternal and fetal outcomes with hypothyroidism and subclinical thyroid dysfunction. Fetal Matern Med Rev 2011;22:169–87.
[6] Anckaert E, Poppe K, Van Uytfanghe K, Schiettecatte J, Foulon W, Thienpont LM. FT4 immunoassays may display a pattern during pregnancy similar to the equilibrium dialysis ID–LC/tandem MS candidate reference measurement procedure in spite of susceptibility towards binding protein alterations. Clin Chim Acta 2010;411:1348–53.
[7] Trajkovic-Arsic M, Müller J, Darras VM, Groba C, Lee S, Weih D, et al. Impact of monocarboxylate transporter-8 deficiency on the hypothalamus-pituitary-thyroid axis in mice. Endocrinology 2010;151:5053–62.
[8] Lim SK, Pilon A, Guéchot J. Biotin interferes with free thyroid hormone and thyroglobulin, but not TSH measurements using Beckman-Access immunoassays. Annales Endocrinol 2017;78:186–7.
[9] Koulouri O, Moran C, Halsall D, Chatterjee K, Gurnell M. Pitfalls in the measurement and interpretation of thyroid function tests. Best Pract Res Clin Endocrinol Metab 2013;27:745–62.
[10] Gurnell M, Halsall DJ, Chatterjee VK. What should be done when thyroid function tests do not make sense? Clin Endocrinol 2011;74:673–8.
[11] Beck-Peccoz P, Persani L. Thyrotropinomas. Endocrinol Metab Clinics N Am 2008;37:123–34.
[12] Dayan CM. Interpretation of thyroid function tests. The Lancet 2001;357(9256):619–24.
[13] Clark P, Gordon K. Challenges for the endocrine laboratory in critical illness. Best Pract Res Clin Endocrinol Metab 2011;25:847–59.
[14] Després N, Grant AM. Antibody interference in thyroid assays: a potential for clinical misinformation. Clin Chem 1998;44:440–54.
[15] Favresse J, Burlacu M-C, Maiter D, Gruson D. Interferences with thyroid function immunoassays: clinical implications and detection algorithm. Endocr Rev 2018;39:830–50.
[16] Chin KP, Pin YC. Heterophile antibody interference with thyroid assay. Int Med 2008;47:2033–7.

Liliana Fonseca [a,*]
Vânia Silva Benido [a]
João Pessanha [b]
Maria Teresa Pereira [a]
Joana Vilaverde [a]
Jorge Dores [a]
[a] *Endocrinology Department, Centro Hospitalar e Universitário do Porto, Largo Professor Abel Salazar, 4099-001 Porto, Portugal*
[b] *Clinical Chemistry Department, Centro Hospitalar e Universitário do Porto, Largo Professor Abel Salazar, 4099-001 Porto, Portugal*

* Corresponding author.
E-mail address: lilianafonsecaa@gmail.com
(L. Fonseca)

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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

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 1.74 mmol/l at the admission. Albuminemia (colorimetric technique) was 41 g/l. Phosphoremia (0.94 mmol/l) and magnesemia (0.82 mmol/l) 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/ml (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 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 6 mg 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 2 g of Calcium hydrochloride/24 h 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 4 g/day and Alfacalcidol 1 μg/day allowing normalization of calcemia 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

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Disclosure of interest

The authors declare that they have no competing interest.

References

[1] Di Filippo L, Formenti AM, Rovere-Querini P, Carlucci M, Conte C, Ciceri F, et al. Hypocalcemia is highly prevalent and predicts hospitalization in patients with COVID-19. Endocrine 2020:1–4.
[2] Di Filippo L, Formenti AM, Doga M, Frara S, Rovere-Querini P, Bosi E, et al. Hypocalcemia is a distinctive biochemical feature of hospitalized COVID-19 patients. Endocrine 2021;71(1):9–13.
[3] Liu J, Han P, Wu J, Gong J, Tian D. Prevalence and predictive value of hypocalcemia in severe COVID-19 patients. J Infect Public Health 2020;13(9):1224–8.
[4] Martineau AR, Jolliffe DA, Hooper RL, Greenberg L, Aloia JF, Bergman P, et al. Vitamin D supplementation to prevent acute respiratory tract infections: systematic review and meta-analysis of individual participant data. The BMJ 2017:356.
[5] Hastie CE, Mackay DF, Ho F, Celis-Morales CA, Katikireddi SV, Niedzwiedz CL, et al. Vitamin D concentrations and COVID-19 infection in UK Biobank. Diabetes Metab Syndr 2020;14(4):561–5.
[6] Grant WB, Lahore H, McDonnell SL, Baggerly CA, French CB, Aliano JL, et al. Evidence that Vitamin D Supplementation Could Reduce Risk of Influenza and COVID-19 Infections and Deaths. Nutrients 2020;12(4.).
[7] Prévention du syndrome de renutrition inappropriée (SRI)des patients Covid 19 [Internet]. [cited 2020 Nov 25]. Available from: https://www.sfncm.org/images/stories/Fiches_Covid_19/Fiche_03_COVID-19.pdf.
[8] Barazzoni R, Bischoff SC, Breda J, Wickramasinghe K, Krznaric Z, Nitzan D, et al. ESPEN expert statements and practical guidance for nutritional management of individuals with SARS-CoV-2 infection. Clin Nutr 2020;39(6):1631–8.
[9] Bossoni S, Chiesa L, Giustina A. Severe hypocalcemia in a thyroidectomized woman with Covid-19 infection. Endocrine 2020;68(2):253–4.
[10] Pla B, Silva M, Arranz A, Marazuela M. Hipocalcemia severa y resistente al tratamiento en paciente con neumonía bilateral COVID-19. Endocrinol Diabetes Nutr 2020.
[11] Elkattawy S, Alyacoub R, Ayad S, Pandya M, Eckman A. A Novel Case of Hypoparathyroidism Secondary to SARS-CoV-2 Infection. Cureus 2020;(8.).
[12] Pal R. COVID-19, hypothalamo-pituitary-adrenal axis and clinical implications. Endocrine 2020:1–2.
[13] Pal R, Banerjee M. COVID-19 and the endocrine system: exploring the unexplored. J Endocrinol Invest 2020:1–5.
[14] Tissue expression of ACE2 - Staining in parathyroid gland - The Human Protein Atlas [Internet]. [cited 2020 Dec 17]. Available from: https://www.proteinatlas.org/ENSG00000130234-ACE2/tissue/parathyroid+gland.

Jean-Baptiste Bonnet [a,*]
Elise Berchoux [a]
Ariane Sultan [b]
[a] *Département d'Endocrinologie, Diabète, CHU de Montpellier, Université de Montpellier, Montpellier, France*
[b] *Département d'Endocrinologie, Diabète, Nutrition Inserm 1411, CHU de Montpellier, Université de Montpellier, Montpellier, France*

* Corresponding author.
E-mail address:
jean-baptiste-bonnet@chu-montpellier.fr
(J.-B. Bonnet)

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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 refeered 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: