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Letter to the Editor

Early detection of the SARS-CoV-2 P.1 variant in Rio Grande do Sul, Brazil: a case report

Fabiana Caroline Zempulski Volpato MSc[1,2,3], Priscila Lamb Wink PhD[1,2,4], Francielle Liz Monteiro PhD[1,2,5], Julia Biz Willig MSc[1,2], Tarsila Vieceli MD[6], Andreza Francisco Martins Ph.D,[1,4,7,8], Alexandre Prehn Zavascki MD, PhD[1,6,9] and Afonso Luís Barth Ph.D[1,2,3,4]

[1]LABRESIS – Laboratório de Pesquisa em Resistência Bacteriana, Hospital de Clínicas de Porto Alegre, Porto Alegre, Rio Grande do Sul, Brasil, [2]Laboratório de Diagnóstico de SARS-CoV-2, Hospital de Clínicas de Porto Alegre, Rio Grande do Sul, Brasil, [3]Programa de Pós-Graduação em Ciências Médicas, Universidade Federal do Rio Grande do Sul, Porto Alegre, Rio Grande do Sul, Brasil, [4]Programa de Pós-Graduação em Ciências Farmacêuticas, Universidade Federal do Rio Grande do Sul, Porto Alegre, Rio Grande do Sul, Brasil, [5]Laboratório de Diagnóstico Molecular, Universidade Franciscana, Santa Maria, Rio Grande do Sul, Brasil, [6]Infectious Diseases Service, Hospital de Clínicas de Porto Alegre, Porto Alegre, Rio Grande do Sul, Brasil, [7]Bioinformatic Core, Hospital de Clínicas de Porto Alegre, Rio Grande do Sul, Brazil, [8]Departamento de Microbiologia, Imunologia e Parasitologia, Instituto de Ciências Básicas, Universidade Federal do Rio Grande do Sul, Brasil and [9]Internal Medicine Department, Universidade Federal do Rio Grande do Sul, Porto Alegre, Rio Grande do Sul, Brazil

To the Editor—In December 2019, a new coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), emerged in China and was associated with coronavirus disease 2019 (COVID-19). Since it was first described, multiple lineages of SARS-CoV-2 have been identified worldwide. Due to the increased transmissibility of the new variants, the initially dominant lineages B.1.1.28 and B.1.1.33 in 2020 in Brazil have been gradually replaced by the variant of interest (VOI) P.2 and by the variant of concern (VOC) P.1.[1] The lineage P.1 was first identified in the beginning of December 2020 in Manaus, Amazonas state, Northern Brazil, and it has been associated with a potential higher transmissibility of the virus.[1,2] In this study, we describe the characteristics of the VOC P.1 from a patient with symptoms in early November 2020 in the Rio Grande do Sul (RS) state, Southern Brazil. This study was approved by the Ethics Committees from Hospital de Clínicas de Porto Alegre (CAAE: 30767420.2.0000.5327).

A middle-aged woman with diabetes mellitus and kidney transplant, a resident of the metropolitan region of Porto Alegre city in Southern Brazil, was admitted to a tertiary-care hospital due to severe COVID-19 on November 29, 2020. The first symptoms of a viral infection were reported 12 days before her hospitalization and included fatigue, dry cough, myalgia, and headache. She had no history of travel and described a direct contact with a person who had been diagnosed with COVID-19 ~16 days before her admission. The COVID-19 worsened, and the patient ultimately died during hospitalization.

On the day of hospital admission, oro/nasopharyngeal swabs were obtained and a nucleic acid amplification testing was carried out to check for SARS-CoV-2 infection. After RNA extraction, the N1 and N2 target genes of SARS-CoV-2 were amplified using a set of primers and probes in a real-time reverse transcriptase polymerase chain reaction (RT-qPCR) as described by the US Centers for Disease Control and Prevention (CDC).[3]

As part of genomic surveillance research that was approved by the Ethics Committee from Hospital de Clínicas de Porto Alegre (CAAE: 30767420.2.0000.5327), the RNA of the clinical specimen (164_LABRESIS) was subjected to whole-genome sequencing (WGS). The RNA of the clinical specimen was extracted using QIAamp Viral RNA Mini Kit (QIAGEN GmbH, Hilden, Germany) to obtain a final elution volume of 60 µL. Sequencing libraries were prepared using the CleanPlex SARS-CoV-2 panel (Paragon Genomics, Hayward, CA) protocol for target enrichment and library preparation according to the manufacturer's instructions (https://www.paragongenomics.com/wp-content/uploads/2020/03/UG4001-01_-CleanPlex-SARS-CoV-2-Panel-User-Guide.pdf). The resulting libraries were built using an Illumina MiSeq sequencer (Illumina, San Diego, CA). Consensus sequences were generated by the QIASeq SARS-CoV-2 pipeline (QIAGEN CLC Genomics Workbench 21, Germantown, PA) and a high-quality whole-genome sequence was obtained (coverage genome: 99.97%; reads Q>30: 628,996; size of genome: 29.89 Kb). The specimen 164_LABRESIS was classified as the P.1 variant using the Phylogenetic Assignment of Named Global Outbreak Lineages (Pangolin) software tool (version 3.0.2),[4] and the sequence was deposited into the GISAID database (https://www.gisaid.org/; no. EPI_ISL_3233232).

According to Pangolin,[4] the VOC P1 is characterized by 17 amino acid changes (including 10 in the spike protein), 3 deletions, 4 synonymous mutations, and one 4-bp nucleotide insertion compared to the most closely related sequence of B.1.1.28 available from April 4, 2020 (GISAID ID: EPI_ISL_722052).[2] The P.1 lineage-defining mutations in the spike protein (especially those at the receptor-binding domains [RBDs] K417T, E484K, and N501Y) raise concern because they may enhance ACE2 affinity and contribute to antibody evasion.[5] The specimen 164_LABRESIS presented 13 of the total of mutations characteristics of the VOC P.1 (orf1ab: S1188L and K1795Q; del: 11288:9; spike: L18F, T20N, D138Y, R190S, K417T, E484K, N501Y and H655Y; orf8: E92K; nucleocapsid:

Author for correspondence: Fabiana Caroline Zempulski Volpato, E-mail: fabiana_volpato@yahoo.com.br

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P80R). The isolate 164_LABRESIS has 9 of 10 mutations in the spike protein, including the 3 P.1 lineage-defining mutations associated with the RBD region.

The first description of the VOC P.1 in Brazil occurred in mid- to late December 2020 in Manaus, Amazon state.[1] According to Faria et al,[1] P.1 comprises 42% of the genomes sequenced in December 2020 in Northern Brazil. However, this lineage was not found in the specimens collected in November 2020 in the same region.[1] In Rio Grande do Sul, the first report of the VOC P.1 occurred on January 29, 2021, from a patient hospitalized in Gramado city.[6] Thereafter, Silva et al[7] reported that the P.1 lineage had already been circulating in the state in late November 2020 but was not disseminated among individuals in the region. The identification of 164_LABRESIS confirms the presence of the VOC P.1 at least in mid-November 2020 in Rio Grande do Sul, according to the onset of symptoms reported 12 days before admission. These findings suggest that the P.1 variant could already have been circulating in Rio Grande do Sul before becoming increasing prevalent in the region. Notably, the predominance of the VOC P.1 in Porto Alegre was documented only in February 2021, when this lineage was associated with a rapid increase in hospitalization rates in the region.[8]

Our findings suggest that the VOC P.1 in a specimen from a patient infected in early November 2020 could already have been circulating in Southern Brazil at least 3 months before the increased predominance of this lineage. In fact, genomic surveillance is a fundamental tool for understanding the evolution and spread of SARS-CoV-2 variants.

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Conflicts of interest. A.P.Z., A.L.B., and A.F.M. are research fellows of the National Council for Scientific and Technological Development (CNPq), Ministry of Science and Technology, Brazil. APZ has received a research grant from Pfizer not related to this work. All other authors have no conflicts to declare.

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