LETTERS

Incidental diagnosis of Fahr's syndrome after coronavirus disease 2019 infection with the fatal outcome

Dear Editor,

Fahr's syndrome is a rare condition with diagnostic criteria including bilateral calcifications of the basal ganglia, progressive extrapyramidal features, young age of onset, and absence of autosomal dominant inheritance[1]. The prevalence of this syndrome occurs in approximately 0.5% of the population[2]. The most common causes of Fahr's syndrome are hypoparathyroidism and pseudohypoparathyroidism. Diagnosis of hypoparathyroidism relies on laboratory data (low calcium, high phosphorous serum levels, decreased or absent parathyroid hormone)[1]. Patients with Fahr's syndrome may be asymptomatic or predisposed to various other symptoms. The most common present movement disorder is Parkinsonism. Clinical manifestations include fatigue, cerebellar dysfunction, speech difficulty, cognitive impairment, seizure activity, and psychiatric symptoms[2]. The most important diagnostic procedure that shows calcifications in the brain is computed tomography (CT). Recent data suggest that coronavirus disease 2019 (COVID-19) may be associated with neurological and psychiatric manifestations, but the mechanism has not yet been determined.

We describe an incidental diagnosis of Fahr's syndrome with hypoparathyroidism in a 62-year-old male patient admitted to the hospital for weakness and fever with a confirmed laboratory diagnosis of COVID-19 infection. He had no family history of movement disorder, cognitive impairment, or young-onset ischemic stroke. The neurological examination documented extrapyramidal signs with cerebellar dysfunction and anarthria. Laboratory tests showed low serum calcium levels (1.26 mmol/l), ionized calcium 0.52 mmol/l, total vitamin D level 25.21 nmol/l, and serum parathyroid hormone level 4.2 pg/mL. Brain CT revealed symmetric calcifications involving the bilateral basal ganglia and other subcortical structures. Infections (TORCH: toxoplasmosis, syphilis, hepatitis B, rubella, cytomegalovirus, herpes simplex), metabolic disorders, intoxications, and vascular and neurodegenerative diseases were excluded as potential etiological factors. According to previous data, he was diagnosed with primary hypoparathyroidism and Fahr's syndrome without a history of total or subtotal thyroidectomy, neck surgery or radiotherapy, congenital syndrome, autoimmune diseases, or congenital disorders. The patient was treated with intravenous calcium gluconate 3000 mg daily, levodopa/benserazide 325 mg, and amantadine 100 mg daily. On the sixth day of the disease, the state of consciousness worsened (Glasgow coma score: five). Due to severe acute respiratory distress syndrome, he was intubated and mechanically ventilated but succumbed on the seventh day of the disease.

The World Health Organization declared a pandemic due to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection in March 2020. Recent data suggests potential neurotropism, but the exact mechanism has not yet been established. Previous research suggests that the COVID-19 infection may potentially initiate or accelerate neurodegeneration. In addition, patients with neurodegenerative disorders have a higher risk of worsening their pre-existing neurological symptoms. In the literature, a 49-year-old male patient with primary brain calcifications had a confirmed COVID-19 diagnosis and presented with seizures, without comorbidities, following cardiac arrest[3]. Demir et al presented a 68-year-old female SARS-CoV-2-positive patient with Fahr's syndrome with generalized tonic-clonic seizure who died on the eighth day of the disease[2].

We report a SARS-CoV2-positive patient with altered mental status, extracerebral and cerebellar symptoms, and rapid lethal outcome. Clinical, laboratory, and neuroimaging data confirmed the diagnosis of incidental Fahr's syndrome associated with hypoparathyroidism. The link between COVID-19 infection mortality and Fahr's syndrome is still unknown. Further research is needed to confirm this relationship.

Keywords: Fahr's syndrome, calcifications, basal ganglia, hypoparathyroidism, coronavirus disease 2019, COVID-19

Conflict of interest
None declared.

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