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.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
Rehabilitation of peripheral facial palsy associated with COVID-19 in a child: A case report

Dear editor

COVID-19 is caused by infection with the novel severe acute respiratory syndrome coronavirus 2 (SARS-Cov-2). Although initially described in China, it assumed worldwide pandemic proportions. Most patients present a mild clinical disease course, consisting of fever, dry cough and dyspnoea, which resolve without specific treatment. However, clinical manifestations vary, and neurological complications have been described in a substantial proportion of patients [3]. Other aetiologies may cause PFP, such as infection with varicella-zoster virus or HIV, Lyme disease, Mycobacterium tuberculosis infection, sarcoidosis or neoplasms [4]. Coronaviruses are also known to have a neurotropic propensity [5], possibly leading to various neurological complications, including peripheral facial paralysis (PFP). However, the pathogenic mechanisms underlying neurological symptoms in COVID-19 are not completely understood. This report presents the first published case of facial palsy in an otherwise healthy child secondary to infection with the novel coronavirus SARS-Cov-2, with reflections on the natural course and the role of physical and rehabilitation medicine in this form of PFP. Thus, PFP may also be a manifestation of COVID-19 and in the current epidemiological context, physicians evaluating patients with facial palsy should exclude infection with SARS-Cov-2 to prevent diagnostic delays and further transmission of the disease. These patients may have a slower recovery and worse prognosis as compared with those with Bell’s palsy. Thus, rehabilitation needs to be initiated promptly, and close follow-up must be assured to identify and address early complications.

This report presents the first case of PFP secondary to infection with the novel SARS-Cov-2 in the paediatric population, a child without other previous diseases, with reflections about the prognosis and role of PRM.

An 11-year-old boy presented to the emergency department with fever lasting 4 days associated with headache, fatigue and occasional cough. History-taking revealed traumatic brain injury when he was 18 months old, without sequelae. His-family history was unremarkable and he was in sixth grade with moderate achievement. Physical examination revealed no abnormalities except bilateral oropharyngeal hyperaemia with posterior rhinorrhoea. Chest X-ray showed no abnormalities, and laboratory findings were normal except for elevated C-reactive protein level (9.6 mg/dl, normally < 0.5 mg/dl). A nasopharyngeal swab was negative for SARS-Cov-2 and cranial CT revealed no acute abnormalities, and laboratory testing continued to show no abnormalities; a nasopharyngeal swab was negative for SARS-Cov-2 and cranial CT revealed no acute alterations. The boy was discharged with an indication for social isolation.

After 10 days, the boy presented again to the emergency department with right face weakness and difficulty closing the eye for the last 3 days. On examination he showed right-sided PFP with asymmetry at rest, incomplete closure of the right eye (4/5 of palpebral excursion, with Bell’s sign), and no active movements in the right muscles. The remainder of the cranial nerve testing was normal, and no other abnormalities were detected on neurological examination. Laboratory testing continued to show no abnormalities; a nasopharyngeal swab was negative for SARS-Cov-2 and cranial CT revealed no acute alterations. The boy was evaluated by an otorhinolaryngologist, who
established a diagnosis of House-Brackmann grade V PFP possibly secondary to COVID-19. An ophthalmologist excluded keratitis and gave recommendations for eye management (diurnal lubrication and nocturnal eye closure). On discharge from the emergency department, the boy was prescribed a course of oral prednisolone for 5 days and was referred to PRM.

At 12 days after hospital discharge, home exercises were taught in the PRM consultation, and the boy started treatments in the hospital PRM department, 3 times per week, that consisted of neuromuscular re-education and facial mimic training with adjuvant cryotherapy and massage. After a month of intervention, the boy showed slightly less facial asymmetry at rest and almost complete eye closure, although with decreased strength, but maintained muscular inactivity in most of the superior and medium face. Because of difficulties in reconciling treatments with school, the parents asked to pursue the intervention in an outpatient community clinic. However, on follow-up at 2 months, the boy did not show improvement, and the intervention resumed in the hospital PRM department. Because of the slow progress with treatments, a cranial MRI was requested, and the boy was referred for evaluation by neuropaediatrics.

Cranial MRI at 3 months revealed adequate thickness and signal emission of the acoustic-facial bundles, but a right posterior insular-opercular lesion related to an area of encephalomalacia associated with the previous traumatic brain injury was also present (Fig. 1). On evaluation by neuropaediatrics, PFP secondary to COVID-19 was assumed, and owing to important weight loss (6 kg in a week), possible muscular involvement was also inferred. Because of complaints of fatigue and asthenia after COVID-19, with 2 episodes of retrosternal pain and palpitations during running, the boy was also observed by a paediatric cardiologist, who also suggested myocardial affection secondary to COVID-19.

At 2.5 months of disease evolution, synkinesis developed, with simultaneous activation of eye (palpebral orbicularis) and mouth (zygomaticus/risorius) muscles. At this stage, rehabilitation was switched to relaxation and synkinesis control techniques, with weekly frequency. After 6 months of rehabilitation, the boy showed only slight improvement, maintaining a House-Brackmann grade IV PFP, with facial asymmetry at rest aggravated by facial expressions. The boy showed less frequent/intense synkinesis and partial activation of all facial muscles with counter-gravity strength except for orbicularis oculi muscles, which showed counter-resistance strength.

Here we describe a case of PFP related to SARS-Cov-2 infection in an 11-year-old boy. A few case reports have described PFP associated with COVID-19 [9–11]; although with no rehabilitation considerations. Only one case was described in children [12], although it refers to a child with previous chromosomal anomaly. Thus, to our knowledge, this is the first report of PFP secondary to COVID-19 in a child without congenital abnormalities.

The diagnosis of PFP is clinical, with additional testing required to exclude a secondary cause. Viral involvement is a presumed aetiology, but in our case, along with a positive nasopharyngeal swab for SARS-Cov-2, possible muscular and cardiac involvement reinforces the association with COVID-19 because these manifestations were previously described [13,14]. The time since the first positive test and the start of neurological symptoms agrees with previous cases [9] and also reinforces the association with COVID-19. Additionally, other secondary causes of PFP were excluded. However, although rather improbable, Bell’s palsy could have been an unrelated event occurring with COVID-19.

Coronaviruses have neurotropic potential, as described in studies demonstrating that severe acute respiratory syndrome and middle east respiratory syndrome coronaviruses could have a transcribial route to the brain [5]. However, the pathogenic mechanisms underlying neurological symptoms in COVID-19 are not completely understood. SARS-Cov-2 likely also possesses potential neurotropism [1,5], entering the central nervous system via the olfactory nerve and bulb [15], causing direct neurological damage owing to its high affinity for angiotensin-convert enzyme 2 receptors, widely expressed in glial cells and neurons [5]. However, the development of PFP in the early phases of COVID-19 with negative PCR results for SARS-Cov-2 in cerebrospinal fluid suggests a parainfectious phenomenon [9]. In our case, lumbar puncture was not performed; however, the results would probably have been negative because a PCR result for SARS-Cov-2 in cerebrospinal fluid has not been described [16]. Thus, an immune-mediated injury by molecular mimicry, from proinflammatory cytokines rather than direct viral neurotropism, may be another mechanism [9].

Imaging studies are not typically indicated in the early evaluation of PFP. However, cranial MRI may show increased thickness and signal emission of the facial nerve with gadolinium enhancement within the first 1 to 2 months after onset. After this time, inflammation decreases and MRI findings may be normal, which explains why the cranial MRI performed at 3 months in our case failed to show abnormalities. To exclude a central cause, MRI is indicated later in the PFP course in case

Fig. 1. Cranial MRI at 3 months in an 11-year-old boy with peripheral facial palsy. (A) Adequate thickness and signal emission of the acoustic-facial bundles without anomalous gadolinium enhancement (arrows). (B) Right posterior cortico-subcortical insular and parieto-opercular lesion related to an area of encephalomalacia with slight regional gliosis (arrow).
of no full recovery of facial function. Electrophysiological tests may be used to determine the severity and prognosis of a peripheral facial nerve lesion. However, in our case, electrophysiological tests were not requested because they would be technically difficult to perform and would cause additional pain in the boy without changing the treatment approach.

Oral steroids and PRM are the mainstays of Bell’s palsy treatment. Corticosteroids (prednisolone 1–2 mg/kg/day) are widely used in the early management (<72 hr) to decrease the incidence of permanent deficits. However in non-severe COVID-19, this treatment may delay virus clearance, worsen respiratory symptoms and promote bacterial superinfection [17]. Corticosteroids have been shown to reduce mortality in severe but not non-severe COVID-19 [18]. However, multiple reports have used corticosteroids without complications in PFP associated with SARS-Cov-2 infection [3,11,12,19]. Intervention by PRM is essential to accelerate recovery and prevent sequelae [7]. No rehabilitation considerations have been published regarding PFP associated with COVID-19. Thus, the treatment approach follows treatment guidelines as for Bell’s palsy. However, as more knowledge becomes available, some interventions might be found counterproductive.

Neuromuscular re-education aims to facilitate muscle activity in functional patterns and suppress abnormal muscle activity that interferes with facial function [7]. Treatment techniques include stimulation techniques, passive support techniques, facial mimicry training, muscle strengthening, muscle relaxation techniques and neuromuscular facilitation [7]. When synkinesis develops, control techniques initiated consist of small amplitude movements while preventing the unwanted contraction [7]. Botulinum toxin injection can also temporarily control synkinesis in PFP [20]. However, its use is controversial in this age group and is generally reserved for refractory cases.

No prognostic factors have been described in PFP related to COVID-19. Thus, factors associated with Bell’s palsy are used to determine prognosis. However, PFP associated with COVID-19 might present a different clinical course. Bell’s palsy has a favourable prognosis, with 80% to 85% of cases showing spontaneous resolution within 3 months [7]. However, at 6 months, it is clearer which patients will have sequelae [20]. The possibility of a complete functional recovery is greater in paediatric cases than in adults [8]. Worse prognosis is associated with complete palsy, Ramsay-Hunt syndrome, secondary PFP and absence of recovery by 3 months [20]. A favourable prognostic indicator is represented by clinical improvement within 3 weeks of onset [7]. In our case, at 3 weeks, the boy still showed inactivity in most facial muscles, which indicated worse prognosis for recovery. The slow progress after 6 months of rehabilitation may also indicate a different course of recovery. For these reasons, a worse prognosis in PFP associated with COVID-19 might be suggested, even with rehabilitation. One case report [9] also showed lack of favourable evolution, but the follow-up was too short (1 week) to draw conclusions. In a case series [19] of 8 patients with House-Brackmann grades II and III PFP, after 30-day follow-up, 3 patients still showed weakness. Thus, published case reports do not describe a sufficiently long follow-up to ensure prognosis in PFP related to COVID-19, and more studies are needed.

In conclusion, we describe a case of a PFP related to SARS-Cov-2 infection in an 11-year-old boy. Such patients might have a worse prognosis and slower recovery than those with Bell’s palsy. Thus, rehabilitation needs to be initiated promptly and close follow-up assured.

**Data Availability**

Data will be made available on request.

---

**Declaration of Competing Interest**

None declared.

**References**

[1] Mao L, Jin H, Wang M, Hu Y, Chen S, He Q, et al. Neurologic manifestations of hospitalized patients with coronavirus disease 2019 in Wuhan, China. JAMA Neurology 2020;77:683–90.

[2] Ellul MA, Benjamin L, Singh B, Lant S, Michael BD, Easton A, et al. Neurological associations of COVID-19. Lancet Neurol 2020;19:767–83.

[3] Peiterson E. Bell’s palsy: the spontaneous course of 2500 peripheral facial nerve palsies of different etiologies. Acta Otolaryngol Suppl 2002;7:4–30.

[4] Evston TJ, Croxon CR, Kennedy PGE, Hadlock T, Krishnan AV. Bell’s palsy: aetiology, clinical features and multidisciplinary care. J Neurol Neurosurg Psychiatry 2015;86:1356–61.

[5] Baig AM, Khaleeq A, Ali U, Syeda H. Evidence of the COVID-19 virus targeting the CNS: tissue distribution, host–virus interaction, and proposed neuropathic mechanisms. ACS Chem Neurosci 2020;11(7):995–8.

[6] Slavkin HC. The significance of a human smile: observations on Bell’s palsy. JADA 1999;130(2):269–72.

[7] Matos C. Peripheral facial paralysis: the role of physical medicine and rehabilitation. Acta Med Port 2011;24:907–14.

[8] Ciorba A, Corazzi V, Conz V, Bianchini C, Aimoni C. Facial nerve paralysis in children. World J Clin Cases 2015;3(12):973–9.

[9] Goh Y, Beh DL, Makmur A, Somani J, Chan AC, Pears & Og-sters: facial nerve palsy in COVID-19 infection. Neurology 2020;95:364–7.

[10] Mehta S, Mackinnon D, Gupta S. Severe acute respiratory syndrome coronavirus 2 as an atypical cause of Bell’s palsy in a patient experiencing homelessness. CJEJM 2020;22(5):608–10.

[11] Figueiredo R, Falcão V, Pinto JM, Ramalho C. Peripheral facial paralysis as presenting symptom of COVID-19 in a pregnant woman. BMJ Case Rep 2020;13:e237146.

[12] Theophanous C, Santoro J, Itani R. Bell’s palsy in a pediatric patient with hyper IgM syndrome and severe acute respiratory syndrome coronavirus 2 (SARS-COV-2). Brain Dev 2021;43(2):357–9.

[13] Chang W-T, Toh HS, Liao C-T, Yu W-L. Cardiac involvement of COVID-19: a comprehensive review. Am J Med Sci 2021;361(1):14–22.

[14] Beydon M, Chevalier K, Tabaa OA, Hamroun S, Delletre A-S, Thomas M, et al. Myositis as a manifestation of SARS-COV-2. Ann Rheum Dis 2020;79:217573. In press.

[15] Bridwell R, Long B, Gottlieb M. Neurologic complications of COVID-19. Am J Emerg Med 2020;28(7):1549.e3–1549.e7.

[16] Espindola OM, Siqueira M, Soares C, Lima M, Leite A, Araujo A, et al. Patients with COVID-19 and neurological manifestations show undetectable SARS-Cov-2 RNA levels in the cerebrospinal fluid. Int J Infect Dis 2020;96:367–9.

[17] Tlayjeha H, Mshibah OH, Enanic MA, Alruxawalid A, Tleyjeh R, Thalibe L, et al. Association of corticosteroids use and outcomes in COVID-19 patients: a systematic review and meta-analysis. J Infect Public Health 2020;13(11):1652–63.

[18] Lim WS, Emberson JR, Matham M, Bell JF, Linsell L, Staplin N, et al. Dexamethasone in hospitalized patients with Covid-19 - preliminary report. N Engl J Med 2021;384(4):693–704.

[19] Lima M, Silva MT, Soares C, Coutinho R, Oliveira H, Alfonso L, et al. Peripheral facial nerve palsy associated with COVID-19. J Neurol 2020;266(5):941–4.

[20] Finsterer J. Management of peripheral facial nerve paralysis. Arch Otorhinolaryngol 2008;265:743–52.

Eduardo Freitas Ferreira*
Diogo Portugal
Nuno Silva
Catarina Peixoto
Catarina Matos
Isabel Pereira
Leonor Prates

*Corresponding author at: Physical Medicine and Rehabilitation Department, Hospital Professor Doutor Fernando Fonseca, Anamorada, Portugal

Physical and Rehabilitation Medicine Department, Hospital Professor Doutor Fernando Fonseca, Amadora, Portugal

E-mail address: eduardo.ferreira@hff.min-saude.pt (E.F. Ferreira).