Relevance of neuroimaging in publications on COVID-19 and stroke☆

Relevancia de la neuroimagen en las publicaciones sobre COVID-19 e ictus

Dear Editor:

The COVID-19 pandemic is a challenge for healthcare systems worldwide, and there has been great interest in describing and explaining the manifestations of the disease. The volume of scientific articles published as a result has challenged journals, editors, and reviewers alike.[1] In the case of neurological diseases and stroke, the pandemic has also stimulated the medical community to publish their experience with COVID-19, with a view to helping professionals across the globe to provide care as quickly as possible. A recent review suggests that while COVID-19 does not increase the risk of stroke, it does considerably increase mortality.[2]

However, all parties involved in scientific publication should seek to guarantee the greatest possible accuracy and rigour, with the ultimate aim of providing the best possible care for our patients. With this goal in mind, we would like to comment on 2 studies recently published in this journal.

The valuable study by Barrios-López et al.[3] presents a series of 4 patients with acute stroke treated at the authors' centre, and provides detailed clinical, laboratory, and imaging data. However, we would like to make some remarks on the neuroimages presented. Figure 1 presents a series of images that the authors describe as a ''cerebral perfusion study.'' However, the images presented are not pre-processed CT images or standard parametric maps, but rather a colour-coded representation resulting from automated post-processing. The authors do not indicate which software or algorithm was used to generate these images; these data are essential for correctly interpreting the images. Furthermore, while the caption refers to ''increased mean transit time,'' the images themselves indicate Tmax and CBF. Essentially, Tmax is used to quantify the volume of brain tissue at risk of infarction or ischaemic penumbra,[4,5] but it should not be mistaken for mean transit time (MMT), a parameter used by software tools or algorithms other than those that the authors presumably used. Improper use of cerebral perfusion terminology may result in incorrect interpretation and application of the data presented. Furthermore, the description of figure 2 is, to say the least, unconventional from a radiological viewpoint: the authors describe image A as an ''infratentorial section,'' but the image shows a large part of the temporal and occipital lobes, which may be confusing for less experienced readers.

Aguirre et al.[6] present an excellent description of an interesting case of contrast-induced encephalopathy following mechanical thrombectomy to treat occlusion of the left middle cerebral artery. In this case, figure 1 provides CT perfusion parametric maps with time to peak and cerebral blood volume data, enabling readers to correctly interpret and understand the case. However, including a key explaining the colours used and their correlation with quantitative volume and time data would have enabled more precise interpretation of the images, since the colour space may be modified during post-processing; assessment and quantification is therefore difficult without a colour scale. Regarding image H (24-h CT scan), the authors note the presence of ''signs of oedema,'' which are not evident, in our opinion. The image does not show white matter hypodensity, loss of grey-white differentiation, or mass effect,[7] which constitute the main signs of cerebral oedema on CT images. A more detailed description of the localisation of contrast extravasation (probably the basal ganglia and cerebral cortex) would have also increased the precision of the radiological data presented and provided more complete data on the pathophysiology of the patient's symptoms.

Our comments are intended to emphasise the importance of radiological images in scientific articles and to underscore the role of neuroradiologists in providing precise, expert descriptions and analyses of these images. Research should ideally be multidisciplinary, and radiologists should therefore participate in studies presenting radiological images.[8,9]

References

1. Matias-Guiu J. El papel del editor de una revista científica durante la pandemia del Covid-19. Neurología. 2020;35:223—5, http://dx.doi.org/10.1016/j.nrl.2020.05.005.
2. Trejo-Gabriel-Galán JM. Ictus como complicación y como factor pronóstico de COVID-19. Neurología. 2020;35:318—22, http://dx.doi.org/10.1016/j.nrl.2020.04.015.
3. Barrios-López JM, Rego-García I, Muñoz Martínez C, Romero-Fábrega JC, Rivero Rodríguez M, Ruiz Giménez JA, et al. Ischaemic stroke and SARS-CoV-2 infection: a causal or incidental association? Neurologia. 2020;35:295—302, http://dx.doi.org/10.1016/j.nrl.2020.05.002.
4. Seker F, Pfaff J, Potreck A, Mundiyanapurath S, Ringleb PA, Bendszus M, et al. Correlation of Tmax volumes with clinical outcome in anterior circulation stroke. Brain Behav. 2017;7:3—7, http://dx.doi.org/10.1002/brb3.772.
5. Galinovic I, Kochova E, Khalil A, Villringer K, Piper SK, Fiebach JB. The ratio between cerebral blood flow and Tmax predicts the quality of collaterals in acute ischemic stroke. PLoS One. 2018;13:1—9, http://dx.doi.org/10.1371/journal.pone.0190811.
6. Aguirre C, Trillo S, Ximénez-Carrillo Á, Vivancos J. Encefalopatía por contraste secundaria a posible daño endotelial tras trombectomía mecánica exitosa. Neurología. 2020;35:336—8.
7. Von Kummer R, Dzialowski I. Imaging of cerebral ischemic edema and neuronal death. Neuroradiology. 2017;59:545—53, http://dx.doi.org/10.1007/s00234-017-1847-6.
8. Luyckx E, Bosmans JML, Broeckx BJG, Ceyssens S, Parizel PM, Snoeckx A. Radiologists as co-authors in case reports containing radiological images: does their presence influence quality? J Am Coll Radiol. 2019;16:526—7, http://dx.doi.org/10.1016/j.jacr.2018.07.035.
9. Crean A. Radiologists: ghosts in the machine? BMJ. 1999;318:952, http://dx.doi.org/10.1136/bmj.318.7188.952.

☆ Please cite this article as: Martínez-Barbero JP, Tomás-Muñoz P, Martínez-Moreno R. Relevancia de la neuroimagen en las publicaciones sobre COVID-19 e ictus. Neurología. 2020;35:709—710.

J.P. Martínez-Barbero*, P. Tomás-Muñoz, R. Martínez-Moreno

Sección de Neuroimagen, Hospital Universitario Virgen de las Nieves, Granada, Spain

* Corresponding author.
E-mail address: jpmbhg@hotmail.com (J.P. Martínez-Barbero).

https://doi.org/10.1016/j.nrleng.2020.07.003
2173-5808/

Guillain—Barré syndrome as the first manifestation of SARS-CoV-2 infection

Síndrome de Guillain—Barré como forma de presentación de la infección por SARS-CoV-2

Dear Editor:

In the current context of the COVID-19 pandemic, the neurological complications of SARS-CoV-2 infection and their pathophysiology are being identified.[1—4] We present a case of Guillain-Barré syndrome (GBS) appearing as a manifestation of COVID-19.

Our patient is a 54-year-old woman with history of arterial hypertension, dyslipidaemia, obesity, obstructive sleep apnoea syndrome, polycystic liver and kidney disease and stage 3b chronic kidney disease, and anterior cervical spondylosis due to disc herniation. She visited the emergency department due to a 4-day history of paraesthesia initially manifesting in the fingertips and subsequently in the tips of the toes, progressively associated with distal weakness. She also reported low-grade fever and vomiting of simultaneous onset, and no diarrhoea or respiratory symptoms.

The neurological examination revealed severe distal weakness in the left hand (0/5 in the extensor digitorum and carpal extensor muscles, 2/5 in the flexor digitorum and carpal flexor muscles, and 2/5 in the interosseous muscles) and mild weakness in the right hand (4/5 in the extensor digitorum and carpal extensor muscles and 4/5 in the interosseous muscles); mild weakness was also observed in the left foot (4/5 in the tibialis anterior, lateral peroneus, and tibialis posterior muscles) as well as dysaesthesia in the tips of the toes and Achilles tendon areflexia.

A lumbar puncture revealed albuminocytologic dissociation; considering the clinical diagnosis of GBS with sensorimotor involvement, we started treatment with intravenous immunoglobulins. Foot weakness worsened during the first 24 hours (3/5 in the tibialis anterior, lateral peroneus, and tibialis posterior muscles bilaterally) but subsequently showed a progressive improvement, with complete resolution at 10 days and only residual dysaesthesia persisting in the tips of the toes.

Given the epidemiological context, SARS-CoV-2 is considered a possible trigger of GBS; a polymerase chain reaction (PCR) test of nasopharyngeal exudate was conducted, yielding negative results. During the course of the symptoms, the patient presented fever, and vomiting persisted, as well as an alveolar infiltrate in the right lower and middle lobes in a chest radiography, with negative results in urine pneumococcal antigen and *Legionella* antigen tests. Blood analysis results revealed lymphocytopaenia, as well as elevated levels of D-dimer, ferritin, and lactate dehydrogenase. A second PCR test for SARS-CoV-2, performed at 24 hours, yielded positive results; therefore, we started empirical treatment with ceftriaxone, azithromycin, hydroxychloroquine, and lopinavir/ritonavir.

At day 6 after admission, the patient presented rapidly progressive severe respiratory insufficiency due to acute respiratory distress syndrome (ARDS), requiring non-invasive ventilatory support with continuous positive airway pressure (CPAP). An additional chest radiography revealed bilateral alveolar opacities. Given the clinical and radiological progression, we decided to administer treatment with methylprednisolone and tocilizumab.

Finally, the patient's respiratory symptoms progressively improved, and we were able to suspend ventilatory support and subsequently oxygen therapy. The patient was discharged 15 days after admission, with no vomiting or neurological symptoms.

In the aetiology study, the autoimmunity study yielded negative results for ANA, ANCA, RF, anti-dsDNA, and antigangliosides and positive results for anti-Ro antibodies; serology studies for cytomegalovirus, *Borrelia*, *Campylobacter*, *Mycoplasma*, HIV, and syphilis all returned negative results. Cerebrospinal fluid PCR testing for SARS-CoV-2 yielded negative results. An electrophysiological study performed 2 months later showed decreased amplitude of sensory potentials in all 4 limbs and, to a lesser degree, decreased motor evoked potential amplitudes; an electromyography using coaxial needle electrodes showed a slightly neurogenic recruitment pattern in distal muscles of the lower limbs, with no signs of denervation. Results were compatible with the acute motor-sensory axonal neuropathy (AMSAN) subtype of GBS, in the recovery phase.

GBS is a paradigmatic post-infectious inflammatory disease, with a known association with such viral infections as influenza, cytomegalovirus, or Epstein-Barr virus; more recently, it has been associated with emerging viruses, such as Zika, dengue, or chikungunya. Other cases have been reported in association with other coronaviruses, such as the Middle East respiratory syndrome (MERS) coronavirus.[4,5]