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Case study

Intervenciones de enfermería aplicadas a una persona con EVC isquémico secundario a infección por SARS-CoV-2

Nursing interventions applied to a person with ischemic CVE secondary to SARS-CoV-2 infection

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Resumen

Introducción: El presente estudio de caso muestra el impacto de la infección por SARS-CoV-2, en particular, sus manifestaciones neurológicas, que incluyen la anosmia, ageusia, adinamia, confusión, alteración en el estado de conciencia y alteración en la circulación cerebral, y que pueden generar secuelas como el evento vascular cerebral (EVC) isquémico.

Objetivo: aplicación de intervenciones y acciones especializadas a un paciente con EVC isquémico secundario a infección por SARS-CoV-2, con base en la teoría del autocuidado.

Material y métodos: se realizó una valoración neurológica del paciente mediante un instrumento de valoración elaborado por estudiantes del posgrado de Enfermería Neurológica con base en los requisitos de autocuidado universal planteados por Dorothea Orem. Se mantuvo un seguimiento holístico, y se proporcionaron cuidados según planes de intervención específicos para las alteraciones presentadas.

Descripción del caso clínico: paciente con diagnóstico médico de EVC isquémico de la arteria cerebral media izquierda. Debido al estado de salud que presentaba durante la aplicación del instrumento de valoración, para la adquisición de datos se contó con el apoyo de la persona responsable, mediante consentimiento informado.

Relevancia: La COVID-19, ocasionada por la infección de SARS-CoV-2, se caracteriza por la afinidad del virus a los receptores ECA2 presentes en todo el organismo humano. El cerebro, debido a los múltiples receptores de este tipo que presenta, genera una sintomatología específica. Esto deriva en cuadros neurológicos que, dependiendo de la gravedad de la enfermedad, podrían ocasionar secuelas en la persona infectada.

Conclusiones: El personal de enfermería tiene un papel importante en el cuidado de las personas infectadas por SARS-CoV-2, ya que mediante los cuidados especializados que brindan posibilitan su estabilización y recuperación.

Palabras clave: SARS-CoV-2, COVID-19, síndrome post agudo de COVID-19, factores de riesgo, trombosis, síndrome de liberación de citoquinas, accidente cerebrovascular.

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Abstract

Introduction: The present case study shows the impact of SARS-CoV-2 infection, in particular, its neurological manifestations, which include anosmia, ageusia, adynamia, confusion, altered state of consciousness, and altered cerebral circulation, and which can generate sequelae such as ischemic cerebral vascular event (CVE).

Objective: application of interventions and specialized actions to a patient with ischemic CVE secondary to SARS-CoV-2 infection, based on the self-care theory.

Material and methods: a neurological assessment of the patient was carried out using an assessment instrument developed by students of the postgraduate course in Neurological Nursing based on the universal self-care requirements proposed by Dorothea Orem. Holistic follow-up was maintained, and care was provided according to specific intervention plans for the alterations presented.

Description of the clinical case: patient with a medical diagnosis of ischemic CVE of the left middle cerebral artery. Due to the state of health presented during the application of the assessment instrument, the data acquisition was supported by the responsible person, through informed consent.

Relevance: COVID-19, caused by SARS-CoV-2 infection, is characterized by the affinity of the virus for ECA2 receptors present throughout the human body. Due to its multiple receptors of this type, the brain generates a specific symptomatology. This results in neurological symptoms which, depending on the severity of the disease, may cause sequelae in the infected person.

Conclusions: Healthcare personnel play an important role in caring for people infected with SARS-CoV-2 since their specialized nursing services enable stabilization and recovery.

Keywords: SARS-CoV-2, COVID-19, post-acute COVID-19 syndrome, risk factors, thrombosis, cytokine release syndrome, stroke.

Introduction

At the end of 2019, the world faced a complex health situation arising from the discovery of a new variant of a virus of the *Coronaviridae family*, which distinguishes itself by its rapid transmissibility and great impact on infected individuals.

COVID-19 is a disease of recent appearance caused by SARS-CoV-2 infection and characterized by the affinity of the virus for the ECA2 receptors present throughout the human body. This particularity, together with the host conditions (immune response, presence of risk factors, disease onset symptoms, management, and follow-up) makes possible the development of sequelae in the infected person.

Due to its multiple ACE2 receptors, the brain generates a specific symptomatology that can manifest in infected persons as anosmia, ageusia, adynamia, allodynia, headache, dizziness, altered state of consciousness, confusion, ataxia and alterations in cerebral circulation. The latter is the consequence of a set of systemic inflammatory phenomena, in

which the host immune response is directly proportional to the severity of the disease. If the inflammatory response is not adequately and timely controlled, changes begin to develop in the cerebral circulation that may generate optimal conditions for the development of an ischemic cerebral vascular event, which is considered a sequela of SARS-CoV-2 infection.

Background

In December 2019, a cluster of severe pneumonia cases with unknown etiology occurred in Wuhan City, China. Two-thirds of the cases had reported visiting the Wuhan seafood market, so on December 30 of the same year, it was closed. The following day, the World Health Organization (WHO) was notified of these cases of pneumonia of idiopathic etiology.[1]

On January 12, 2020, the genome of the new virus was analyzed and named 2019-nCov.[1] Subsequently, on February 11, the disease was taxonomically designated as SARS-CoV-2 (SARS-CoV-2), and then named coronavirus disease 2019 (COVID-19).2 Following the presence of cases of this coronavirus in countries such as Thailand, Japan, Korea, Spain, and 109 other countries, on March 11, 2020, the WHO declared a global public health emergency due to the COVID-19 pandemic.[1,2]

The term coronavirus derives from the corona-like appearance of the virus envelope, observable by electron microscopy. It is a virus with single-stranded RNA and a great genetic diversity, which makes it easily recombine and generate new types, some capable of infecting humans.[1] Coronaviruses belong to the family *Coronaviridae*, subfamily *Orthocoronavirinae*, which consists of four genera (alpha, beta, delta, and gammacoronavirus),[3] and can be classified as mild or highly pathogenic, depending on the clinical picture they cause.[4]

According to its viral structure, it is composed of four proteins: the surface protein (S, class I trimeric fusion glycoprotein), the envelope protein (E), the matrix protein (M), and the nucleocapsid protein (N). Protein S plays an important role in determining tissue tropism in the host.[5]

It has been suggested that the high invasiveness of SARS-CoV-2 into the human body is due to the binding of the virus to an angiotensin II-converting enzyme receptor (ACE2), whose target receptor is located in the lungs, but is also present in the central nervous system, kidneys and other organs. The invasion cycle begins when the S protein of SARS-CoV-2 binds to the ACE2 receptors, causing a structural alteration of the S protein, which facilitates the binding of the viral envelope to the cell membrane and thus the release of the viral RNA into the host cell.[1,6,7]

Neurotropism is the ability of a pathogen to invade and survive in the nervous system.[8] In SARS-CoV-2 infection, it has been reported in dopaminergic neurons, astrocytes, oligodendrocytes, ventricles, medial temporal gyrus, posterior cingulate cortex, and olfactory bulb.[5,8]

There are several hypotheses about the access of SARS-CoV-2 to the central nervous system (CNS) through the following pathways:

- Direct infection: genetic material of the virus has been detected in tissue samples from the nervous system, suggesting that the virus directly invades the nervous system through the ECA2 receptors present in the nervous system.[8]
- Hematogenous: the blood-brain barrier

is composed of vascular endothelium, astrocytes, pericytes, and extracellular matrix, with the vascular endothelium being the structure that hosts ACE2 receptors.[1,6,7,8] Once the virus enters the blood circulation, cytokine production occurs, which generates an increased permeability of the blood-brain barrier and promotes viral entry into the CNS.[8]

- Neuronal: it is the most important mechanism by which neurotropic viruses enter the CNS. Its access occurs through the olfactory or trigeminal nerve, where it spreads locally through the cribriform plate of the ethmoid bone.[1,6,7] Subsequently, it lodges in the brain parenchyma and cerebrospinal fluid, causing an inflammatory and demyelinating reaction.[8]
- Digestive: Viral replication in the intestine causes cell necrosis, inflammation, and dysbiosis, resulting in a breakdown of the intestinal barrier, which allows the virus to pass directly into the blood or lymphatic circulation.[9]

The clinical manifestations present differently in each individual due to their particular characteristics, among which the following stand out:

- Risk factors: comorbidity with diabetes mellitus, hypertension, chronic renal failure, chronic obstructive pulmonary disease (COPD), cancer, heart disease, obesity, overweight, smoking, hypercholesterolemia, hypertriglyceridemia, sedentary lifestyle, poor access to health services, previous vascular disease (aneurysms), age over 60.[10,11]
- Interaction of the virus with ACE2 receptors: ACE2 is present in several cells of the human body, and is responsible for converting angiotensin I into angiotensin II and obtaining vasodilator, antifibrosis, anti-inflammatory, and natriuresis-promoting effects.[12]
- Innate immune response: this is responsible for activating an antiviral response (interaction of macrophages, neutrophils, dendritic cells, and epithelial cells of the target organ), limiting virus replication, and favoring the adequate adaptation of the host immune response.[8,10]

When installed in the CNS, SARS-CoV-2 can trigger neurological manifestations, which by their characteristics are grouped as follows: [4]

1) Neurological conditions secondary to the infectious process, such as ischemic CVE, as well as conditions associated with ACE2 receptor affinity, which can cause hemorrhagic CVE.
2) Neurological symptoms associated with immunosuppression and opportunistic diseases.
3) Neurological symptoms typical of critically ill patients, especially muscle dysfunction or rhabdomyolysis.
4) Neurological symptoms triggered by viral invasion in neurological or muscular areas, which generate olfactory and gustatory alterations, or myositis.
5) Unclassifiable neurological pictures due to lack of information; the presence of hallucinations has been suggested.

Patients with critical COVID-19 develop severe coagulopathy (COVID-19-associated coagulopathy), resulting from the systemic inflammatory response, showing elevated D-dimer levels, thrombocytopenia and pathological data of microangiopathy.[1,8]

Likewise, after a severe infectious disease, such as COVID-19, there is a prolonged and exhausting *compensatory anti-inflammatory*

response syndrome (CARS), which leads to post-infectious immunosuppression, the aim of which is to counteract the proinflammatory state, prevent maladaptive multiorgan dysfunction and restore immune homeostasis.[13] If this response is repressed too much, a stage of prolonged immunosuppression, *immunosuppression, and catabolism syndrome* (PICS) can be reached, in which the recovered patient is more prone to reactivation of the latent SAR-CoV-2 virus, presenting relapses or reactivation. Some authors refer to these relapses and their symptomatology as post-COVID syndrome, long or persistent COVID.[13]

To unify criteria, the National Institute for Health and Care Excellence proposes the following symptomatic phases of SARS-CoV-2 infection:[14]

- Acute COVID-19: duration of up to 4 weeks from symptom onset.
- Post-acute COVID-19: symptomatology persisting for more than 4 weeks or occurrence of late or long-term complications. Includes patients with persistent COVID-19 (long COVID) and post-COVID-19 sequelae.

These sequelae include symptomatology of long duration (months or years), in the absence of any active infection. Its pathophysiology is associated with a state of chronic hyperinflammation, and the following hypotheses have been suggested:[14]

1) There is an alteration in the integrity of the blood-brain barrier, which allows permeability of neurotoxic substances; this, added to the increase in IL-6 levels, exacerbates muscle loss, causing fatigue and muscle weakness.
2) Autoimmune: autoantibodies act on immune system modulator proteins, altering their function and impairing virological control.
3) Persistence of a hypercoagulable state associated with SARS-CoV-2 infection (thromboinflammatory state).
4) Affection of the autonomic nervous system caused by the virus, resulting in orthostatic intolerance syndromes (orthostatic hypotension, vasovagal syncope, and postural orthostatic tachycardia syndrome).
5) Viral persistence caused by a weak or absent immune response (relapses or reinfections).

When an excessive and poorly regulated immune cell response is generated, a cytokine storm is produced that can lead to altered vascular permeability, coagulopathy, and a proinflammatory effect. These conditions may result in cerebral edema.[1,3] It has been suggested that this cytokine storm acts as a causal factor of CVD, as it is associated with intravascular alterations that cause an interruption in cerebral circulation and generate stimulation of the sympathetic nervous system, predisposing to stress cardiomyopathy and cardiac arrhythmias and promoting intracardiac thrombus formation, raising the risk of cardioembolic type CVE.[15]

Some studies propose a relationship between the presence of CVE and certain risk factors, such as age over 60 years, comorbidity of diabetes mellitus, obesity, arterial hypertension, SARS-CoV-2 infection of critical presentation, blood hypercoagulability, hyperlipidemia, coronary artery disease, congestive heart failure, atrial fibrillation, history of CVE or transient ischemic attack, and active smoking.[2,15] Ischemic CVE is characterized by the functional loss of some area of the central nervous system due to thrombotic occlusion

of a cerebral artery; this occlusion occurs suddenly and has the possibility of being transient or permanent.[16]

Normally, cerebral metabolism maintains a balance between cerebral blood flow (CBF), blood pressure, and the blood-brain barrier, the latter two being responsible for the regulation of cerebral blood flow.[17] The cerebral blood flow demand is high and represents 20% of cardiac output. The CBF of an average human adult is 50mL/100gr of tissue/min, in white matter it is 20mL/100gr of tissue/min and in gray matter, it is 80mL/100gr of tissue/min.18 When the CBF begins to decrease, various cellular processes occur: with a CBF of 50-25mL/100gr/min there is a selective loss of neurons; with a CBF of 50-35mL/100gr/min protein synthesis is reduced and there is selective gene expression; with a CBF of 35-25mL/100gr/min the cerebral metabolic rate of glucose rises and then falls, generating anaerobic glycolysis and increased lactate levels; with a CBF of 30-20mL/100g/min the pH decreases and glutamate is released; with a CBF of 25-15mL/100g/min phosphocreatine is reduced and adenosine triphosphate (ATP) depletion and infarction occur; with a CBF of 10mL/100g/min or less anoxic depolarization occurs, intracellular calcium increases, extracellular potassium is released and loss of cellular ionic homeostasis occurs.[18]

The risk factors for CVD are similar to those involved in the development of COVID-19, so it is not unlikely to find people who present COVID-19 and CVD simultaneously.[19] Moya-Guerrero suggests different hypotheses to understand the origin of CVD in people with COVID-19: 19

- Classic arrhythmogenic mechanism: when a person develops a severe infection such as COVID-19, he/she presents a state of stress, which implies an exaggerated sympathetic activation that leads to cardiac arrhythmias, the most frequent being atrial fibrillation, related to the development of ischemic events.
- Inflammatory markers: fibrinogen, C-reactive protein and leukocytes alone are predictive of the course of an ischemic CVE.
- Hypercoagulable state: individuals with COVID-19 have elevated D-dimer levels and a lengthened prothrombin time, resulting in a hypercoagulable state. D-dimer values are generally related to the severity of the disease.
- Hypoxia: firstly, it acts as a prothrombotic factor, since it induces the secretion of cytokines and catecholamines, generating endothelial damage and an alteration in capillary flows. On the other hand, a prolonged hypoxic state causes the secretion of erythropoietin, which produces secondary polycythemia and generates an increase in blood viscosity, slowing vascular flow and thus facilitating the formation of blood clots.
- Endothelial dysfunction: endothelial cells contain ACE2 receptors, a target receptor of SARS-CoV-2, which represents a factor for endothelial dysfunction. This condition facilitates platelet and coagulation factor activation.

Material y methods

A person with a diagnosis of ischemic CVE of the left middle cerebral artery admitted to the fourth-floor hospitalization service of the Manuel Velasco Suárez National

Institute of Neurology and Neurosurgery was selected. The selection was made during the clinical practices of the Neurological Nursing specialty of the morning shift (7:30 am to 2:00 pm), comprised in the period from May 3 to 7, 2021.

Due to the clinical condition of the selected person, the responsible family member was contacted to obtain his or her authorization to carry out the case study, as well as a follow-up of the patient's health status. Authorization was obtained by signing an informed consent form.

The patient was evaluated using a neurological nursing assessment instrument based on the self-care requirements proposed by Dorothea Orem. We had the support of the person responsible for the collection of the missing information. Likewise, the patient's electronic clinical record was accessed, following the specifications dictated on the clinical record of the Electronic health record information system NOM-024-SSA3-2012.Subsequently, a review and analysis of references published nationally and internationally were carried out using databases such as SciELO, PubMed, Medline, Google Scholar, and different websites. Various descriptors were used with the Boolean operators AND and OR, and the keywords previously described were used.

Presentation of the case

SThe patient was Mr. J.D.R., who had a diagnosis of ischemic CVE of the left middle cerebral artery. The patient was administered an assessment instrument based on the universal self-care requirements developed by students of the Neurological Nursing postgraduate course.

Table 1. Health history of Mr. J.D.R. according to the neurological nursing assessment instrument.

HFH	Father deceased of unknown cause with a diagnosis of diabetes mellitus, mother deceased of unknown cause, and a brother with a diagnosis of systemic arterial hypertension.
NPPH	Onset of smoking in adolescence with an approximate consumption of 2 cigarettes per week, suspended after infection by SARS-CoV-2. Occasional alcoholism, drug addictions denied and complete vaccination.
PPH	Allergic to ampicillin, diagnosed with diabetes mellitus in 2014 with no control or follow-up of the disease. Family members reported that he went to private medical consultation only in case of discomfort; sometimes it was necessary to administer rapid-acting insulin, but then the treatment was not continued. Infection by SARS-CoV-2 in December 2020, presenting symptoms with fever, anxiety, adynamic, periods of diaphoresis, and hyperglycemia (capillary glycemia figures were not specified). Treatment was performed at home with supplemental oxygen support through a reservoir mask; NPH insulin, 10 IUI every 24 hours SC; enoxaparin, 40mg every 24 hours SC; protec aspirin, 100mg every 24 hours and relative rest. In addition, the primary caregivers report that after the acute period of COVID-19, pulmonary exercises were started using an incentive inspirometer.

Note: HFH = heredofamilial history, NPPH = nonpathological personal history, PPH = pathological personal history.
Source: Own elaboration.

Health status (update)

On May 5, 2021, the patient presented vital signs of HR 95x', RR 26x', BP 120/80 mmHg, temperature 36.3°C, isochoric pupils, with response to light stimulus and pupillary diameter of 3mm, minimally conscious state, Glasgow scale score of 9, FOUR scale score of 11, oxygen saturation 96%, VAS scale not assessable. Left fronto-temporo-parietal surgical wound with bulging encephalic mass. Motor aphasia, right facial paralysis of central type. Tracheostomy with ventilatory support in CPAP mode, with a FiO2 at 35%, and PEEP of 5. Presence of abundant thick yellowish secretions, peripheral venous access in right thoracic limb with continuous infusion of isotonic solution at 20mL/hour. Upper and lower extremities with skin integrity, right hemiplegia and left hemiparesis, with placement of gentle restraint of the left upper limb because the person was trying to remove the invasive therapeutic devices. Depressible soft abdomen with the presence of a gastrostomy tube with a diet of intakes every 8 hours. Stage II pressure lesion in the sacral region covered with local treatment (skin protector, hydrocolloid dressing, hypocellular dressing, and with permanent secondary dressing of Hipafyx, which was changed at detachment or at one week, whichever occurred first), presence of bladder catheterization with continuous derivation. The patient had periods of diaphoresis due to body mobilization and aspiration of tracheal secretions. Blood tests showed: Na 190 mmol/L, K 4.3 mmol/L, Cl 138.8 mmol/L, glucose 126 mg/dL, urea 36.8 mg/dL, BUN 17 mg/dL, CrS 0.4 mg/dL, uric acid 2.8 mg/dL, cholesterol 64 mg/dL, alkaline phosphatase 225 IU/L, direct bilirubin 0.2 mg/dL, glutamic oxalacetic transaminase 152.2 IU/L, glutamic pyruvic transaminase 179.1 IU/L, serum gamma-glutamyl transferase 94 IU/L, total protein 6.8 g/dL, PT 16.5 sec, INR 1.24, TTP 34.5 sec, leukocytes 11.4, erythrocytes 3.11, hemoglobin 7.9g/dL, hematocrit 26.9%, platelets 339, lymphocytes 7.5%, monocytes 4.5%, eosinophils 2.2%, basophils 0.2%, neutrophils 9.7 and C-reactive protein 4 mg/dL.

Ethical considerations

The professional ethics of nurses should be guided by the fundamental principles of bioethics, which are autonomy, beneficence, nonmaleficence, and justice.[20]

As part of the code of ethics, the International Council of Nurses emphasizes its primary duties, which include promoting health, preventing disease, contributing to the restoration of health and alleviating suffering, and at all times showing respect for human and cultural rights, as well as the right to life, free choice and dignity of individuals.[20]

Often, nurses work with patients who are unable to communicate verbally, which does not imply that they lose their humanity, but rather that they are at a disadvantage.[20]

Neurological nursing assessment

Neurological nursing assessment is a useful method for detecting and recognizing neurological alterations in a person. It is very useful for nursing professionals, since it enables them to detect neurological deterioration in advance, facilitating the identification of possible secondary damage and its timely treatment.[21] The neurological assessment of Mr. J.D.R. is described below.

- Vital signs: heart rate 95 beats per minute, respiratory rate 26 breaths per minute,

blood pressure 120/80 mmHg, body temperature 36.3°C, oxygen saturation 96%, VAS scale not assessable.

- Consciousness: minimally conscious state, with a score of 9 on the Glasgow scale and 11 on the FOUR scale.
- Pupillary assessment: symmetrical pupils with response and diameter of 3mm.
- Cranial nerve assessment: I: not assessable; II: campimetry not assessable. Visual reflexes: photomotor and consensual, present in both eyes; accommodation: not assessable; corneal: present in both eyes; pupillary cutaneous: present in both eyes; III, IV, VI: eye movements, levoversion, dextroversion, supraversion and infraversion, present; little cooperation from the person; V: V1: right portion, corneal reflex present, nasal cavity moistened; V2: not assessable; V3: sensory function not assessable. Presence of jaw movements; VII: motor function: eye closure movements against the examiner's resistance (only movement assessable, when touching the eyelids, the person closes them tightly), sensory function not assessable, secretory function with slightly decreased wetting of both eyes, same as in both nares; VIII: vestibular function not assessable, cochlear function not assessable; IX, X: motor function: the person is not able to swallow, parasympathetic secretomotor function with adequate salivary production, sensitive function with presence of gag reflex; XI not assessable; XII not assessable.
- Assessment of the motor system: the person presents problems with mobilization, as he has right hemiplegia (0/5 on the Daniels muscle strength scale) and left hemiparesis (2/5 on the Daniels muscle strength scale).
- Sensitivity assessment: in the right hemibody the extremities are areflectic and in the left, hyporeflectic (+).

Intervention plan

During the assessment, eight universal self-care requirements were found to be altered. From this, the following diagnoses were developed.

Table 2. Nursing diagnoses made to Mr. J.D.R. according to the self-care requirements that were altered.

Maintenance of sufficient air supply.	• Impaired spontaneous ventilation R/T bulbar center dysfunction in the dorsal respiratory and ventral respiratory nuclei M/B inability to perform diaphragmatic activity during inspiration and expiration, dependence on artificial airway, respiratory muscle fatigue, respiratory rate of 26 per minute. • Ineffective airway patency R/T cranial nerve X alteration, inadequate secretion management M/B altered state of consciousness (minimally conscious state), inability to expectorate thick secretions, presence of rales, increased secretion production.
Maintenance of sufficient food intake.	• Impaired swallowing R/T impairment of the IX and X cranial nerve, altered state of consciousness (minimally conscious state) M/B weakness of lip closure, impaired tongue tone, strength and mobility, fractionated swallows.
Maintenance of a sufficient water supply.	• Hyperosmolar imbalance R/T damage to the supraoptic nucleus of the hypothalamohypophyseal fasciculus M/B excess extracellular solutes (serum sodium 190 mmol/L, chloride 138.8 mmol/L), effective osmolarity 387 mOsm/kg, decreased skin turgor, tachycardia (120 bpm), periods of restlessness.

Provision of care associated with disposal processes.	• Impaired bowel function R/T cranial nerve X dysfunction, prolonged bed rest M/B decreased peristaltic movements (6 per minute), decreased number and frequency of bowel movements (1 time per week), Bristol 1 stool.
Maintaining a balance between activity and rest.	• Impaired physical mobility R/T decreased cerebral blood flow in primary and secondary motor areas (Brodmann areas 4 and 6) M/B right hemiplegia, fatigue, right hemibody Daniels scale score of 0, left hemiparesis, left hemiparesis, left hemibody Daniels scale score of 2. • Loss of total right sensation R/T left parietal lesion (decreased cerebral blood flow in Brodmann's areas 1, 2 and 3) M/B loss of position sense, tactile localization and discrimination, weight sensation, and stereognosis. • Intolerance to mobilization R/T disruption of cerebral blood flow in primary and secondary motor areas (Brodmann areas 4 and 6) M/B tachycardia (132 beats per minute), tachypnea (34 breaths per minute), diaphoresis, restlessness following postural changes, fatigue. • Impaired skin integrity R/T prolonged stay in bed M/B pressure exerted from bony prominence on the skin, intolerance of the person to postural changes, stage II pressure injury in sacral region, Braden scale score of 10.
Maintaining a balance between social interaction and solitude.	• Impaired cognitive function (language) R/T decreased cerebral blood flow in Brodmann areas 44 and 45 M/B motor aphasia, alexithymia, and absence of articulate sound.
Prevention of hazards to human life, functioning, and well-being.	• Incapacity for self-care R/T interruption of cerebral blood flow in primary and secondary motor areas (Brodmann areas 4 and 6) M/B absence of right hemibody contraction, inability to perform self-care independently, Barthel index score of 0. • Disturbance of well-being R/T interruption of cerebral blood flow in left cerebral hemisphere M/B tachycardia (132 beats per minute), diaphoresis, tachypnea (34 breaths per minute). • High risk of tissue hypoxemia R/T hemoglobin levels of 7.9 g/dL. • Risk of bronchial aspiration R/T minimally conscious state, inability to expectorate secretions alone, presence of thick secretions. • Risk of arterial hypertension R/T sodium retention (serum sodium levels of 190 mmol/L), inadequate functioning of the renin-angiotensin-aldosterone system.
Promotion of human functioning and development in social groups in accordance with human potential, known human limitations, and the human desire to be normal.	• Spiritual suffering R/C chronic disease state M/P minimal cooperation with care provided, periods of anger. • Alteration of family processes R/T illness of one of the family members M/B anguish, sadness, and inadequate family adaptation.

Source: own elaboration.

The nursing diagnoses made were reviewed and the intervention plan shown below was generated.

Table 3. Intervention plan of nursing diagnosis 1 applied to Mr. J.D.R.

Altered requirement: maintenance of sufficient air supply.	
Diagnosis: impaired spontaneous ventilation R/T bulbar center dysfunction in the dorsal respiratory and ventral respiratory nuclei M/B inability to perform diaphragmatic activity during inspiration and expiration, dependence on artificial airway, respiratory muscle fatigue, respiratory rate of 26 per minute.	
Nursing system: fully compensatory.	**Objective:** to avoid fatigue in the respiratory musculature, and to maintain a respiratory frequency between 18 and 22 breaths per minute.
Nursing interventions	Nursing actions
Ventilation aid	• Maintain a patent airway. • Position the person to facilitate ventilation/perfusion matching. • Monitor the effects of position change on oxygenation (arterial blood gas levels). • Observe respiratory muscle fatigue.
Airway suction	• Determine the need for tracheal suctioning. • Auscultate breath sounds before and after suctioning. • Provide universal precautions. • Hyperoxygenate with 100% oxygen using the ventilator. • Use sterile disposable equipment for each tracheal suctioning procedure. • Observe the person's oxygen status (SvO2 levels) and hemodynamic status (MAP level and heart rate) immediately before, during, and after suctioning. • Aspirate the oropharynx after completion of tracheal suctioning. • Clean the area around the tracheal stoma after completion of tracheal suctioning.
Management of invasive mechanical ventilation	• Monitor conditions that indicate the need for ventilatory support (respiratory muscle fatigue, neurological dysfunction, anesthesia, refractory respiratory acidosis). • Control activities that increase O2 consumption (fever, pain, or basic nursing activities) that may replace ventilator support settings and cause O2 desaturation. • Provide care to alleviate the person's distress (positioning, tracheobronchial clearance, bronchodilator therapy, sedation and/or analgesia, frequent equipment checks). • Perform aseptic technique in all aspiration procedures. • Monitor the individual's progress on current ventilator settings and make appropriate changes per physician's orders.

Evaluation: the patient maintained oxygen saturation above 96%, reducing episodes of tachypnea and diaphoresis, mechanical ventilation parameters continued under the same conditions (CPAP mode, FiO2 35%, PEEP 5).

Source: own elaboration.

Table 4. Intervention plan of nursing diagnosis 2 applied to Mr. J.D.R.

Altered requirement: maintenance of sufficient water supply.	
Nursing diagnosis: hyperosmolar imbalance R/T damage to the supraoptic nucleus of the hypothalamohypophyseal fasciculus M/B excess extracellular solutes (serum sodium 190 mmol/L, chloride 138.8 mmol/L), effective osmolarity 387 mOsm/kg, decreased skin turgor, tachycardia (120 bpm), periods of restlessness.	
Nursing system: fully compensatory.	**Objective:** to restore the water-solids balance.
Nursing interventions	Nursing actions
Electrolyte management	• Observe if serum electrolytes are abnormal. • Maintain permeable IV access. • Administer fluids as prescribed. • Maintain adequate intake and elimination records. • Obtain samples for laboratory analysis of electrolyte levels (arterial blood, urine, and serum gases). • Place cardiac monitor. • Control electrolyte imbalances associated with hypernatremia (hyperchloremia and hyperglycemia). • Look for signs of dehydration (decreased sweating, decreased urine output, decreased skin turgor, and dry mucous membranes). • Promote skin integrity.
Liquid handling	• Keep an accurate record of intake and elimination. • Perform bladder catheterization. • Monitor hydration status (moist mucous membranes, adequate pulse, and orthostatic blood pressure). • Monitor vital signs. • Administer IV therapy as prescribed.
Electrolyte monitoring	• Monitor serum electrolyte levels. • Observe signs and symptoms of hypernatremia: extreme thirst, fever, dryness, sticky mucous membranes, tachycardia, hypotension, lethargy, confusion, altered mental status, and seizures. • Observe signs and symptoms of hyperchloremia: weakness, lethargy, rapid, deep breathing, and coma.
Evaluation: the person achieved a correction in serum electrolyte levels and effective osmolarity (Na 139 mmol/L, Cl 99.6 mmol/L, and 284.7 mOsm/kg).	

Source: own elaboration.

Table 5. Intervention plan of nursing diagnosis 3 applied to Mr. J.D.R.

Altered requirement: maintenance of a balance between activity and rest.	
Nursing diagnosis: loss of total right sensory R/T left parietal lesion (decreased cerebral blood flow in Brodmann's areas 1, 2, and 3) M/B loss of position sense, tactile localization and discrimination, weight sensation, and stereognosis.	
Nursing system: fully compensatory.	**Objective:** to diminish the effects of the deficit in perception and prevent related complications.

Nursing interventions	Nursing actions
Skin stimulation	• Select the type of skin stimulation best suited to the person and his/her conditions (massage, cold, heat, menthol, vibration, or transcutaneous electrical nerve stimulation). • Apply stimulation directly on or around the affected site. • Establish the duration and frequency of stimulation according to the method chosen.
Skin surveillance	• Monitor skin color and temperature. • Observe for areas of discoloration and loss of skin integrity. • Observe areas of friction and pressure. • Observe for skin rashes or abrasions. • Observe excessive moisture or dryness of the skin. • Take note of skin changes. • Implement measures to prevent further deterioration (alternating pressure cushion, repositioning schedule). • Use an assessment tool to identify individuals at risk of loss of skin integrity (Braden scale).
Environmental management	• Create a safe environment for the person. • Identify the individual's safety needs based on physical function, cognitive, and behavioral history. • Provide a clean and comfortable environment. • Avoid exposing the skin to irritants. • Identify environmental safety hazards (physical, chemical, and biological). • Eliminate risk factors from the environment when possible.

Evaluation: the person showed a relaxed facial expression and posture for longer periods. There were no lesions associated with right total hemianesthesia.

Source: own elaboration.

Discharge plan

The discharge plan provides primary caregivers, nursing staff, and the family with information on the care to be provided once the patient leaves the hospital and goes home, to avoid complications, reduce possible hospital readmissions and, in general, improve the patient's quality of life.[22]

The following is a summary of the activities used in the discharge plan for Mr. J.D.R. based on the CUIDARME mnemonic (communication, urgency, information, diet, environment, recreation and use of free time, medications and treatments, spirituality):

Table 8. Discharge plan for Mr. J.D.R. based on the CUIDARME mnemonic

Effective communication	With the support of the nursing consultancy service, scheduled training was provided to caregivers before Mr. J.D.R.›s discharge, and relevant information was provided; the link with nutrition and dietetics, palliative care, and rehabilitation services was facilitated.
Emergencies and warning signs	Primary caregivers are told that if their family member presents with any of the following signs, they should go to the hospital nearest to their home: drowsiness for abnormal periods, persistent headache, vomiting, seizures, confusion or disorientation, generalized pain that does not decrease with medication, new pressure lesions, increased secretions, pain or swelling in the pressure lesion that was present in the sacral region, constipation, increased tracheal secretions and changes in their characteristics, presence of secretions around the stomas (tracheal and gastric), pain or erythema, or sensation of shortness of breath.

Information	Caregivers were trained on hand washing techniques, airway management, gastrostomy tube care, skin care, prevention of pressure injuries, treatment of stage II pressure injury in the sacral region of Mr. J.D.R., prevention of constipation, mobilization in the environment, sponge bath, use of the communication board.
Diet	The nutrition and dietetics service explained to Mr. J.D.R.'s caregivers the preparation of food, the amounts to be administered, and the intervals at which they would be administered. The nursing staff oriented them on the recommendations to promote feeding (positioning of the person at the time of feeding, speed of infusion of the diet, temperature of the diet), care of the gastrostomy tube (washing the tube after the administration of food or medication, avoiding traction of the tube), and the importance of hydration and oral hygiene.
Environment	Based on the background information provided regarding the person's environment, guidance was given on how to maintain adequate lighting in the room intended for Mr. J.D.R., hygienic measures for the family member and his environment, the need to remove objects that could obstruct the passage and the use of bed rails.
Recreation and use of free time	The person's physical and occupational rehabilitation was carried out by INNN's rehabilitation service. The nursing staff guided the caregivers on the importance of maintaining body alignment, modifying postural reactions, avoiding overstimulation, taking walks near the home, as well as providing Mr. J.D.R. with readings, movies, or music that are of interest to him.
Medications and harmacological treatments	The caregivers were oriented on the indications of the medications prescribed for Mr. J.D.R., their possible adverse effects, the most appropriate schedules for their administration according to his rhythm of life and the characteristics of the medications, the routes of administration, the care required for the conservation of the medications and on strategies for polypharmacy (use of pill dispensers, schedules, and calendars, alarms). Also, with the support of the nursing consultancy service, the primary caregivers were educated on the characteristics of the cures to be performed on Mr. J.D.R.'s pressure injury, the frequency and the products to be used.
Spirituality	It was recommended that caregivers continue to strengthen their religious beliefs (attendance at ceremonial meetings) and involve Mr. J.D.R. to promote relief. Also, always to facilitate the expression of each family member's feelings.

Source: own elaboration.

Relevance

A fundamental aspect of increasing the generation of evidence by nursing professionals is to encourage and guide them to engage in evidence-based research and publication strategies. It is only through this exercise and familiarization with best practices that the importance of specialized care will be magnified, thereby achieving greater recognition for professionals and the discipline. In short, this will have a positive impact on the interventions provided to the population on which the activities of the nursing staff are focused.

Conclusions

This case study exemplifies the importance of the control and management of risk factors present in the population (comorbidities), as well as the relevance of timely and adequate care of people infected with SARS-CoV-2, as it is a prognostic factor for their health status.

It is gratifying to report the successful application of interventions and actions according

to the self-care theory to Mr. J.D.R., who was admitted to INNNN with an ischemic cerebral vascular event following infection by SARS-CoV-2. Despite the constant challenges during the patient's clinical follow-up, the objectives set were met through the support of the multidisciplinary team, and the participation of Mr. J.D.R. and his family, who proved to be an excellent pillar in his clinical evolution.

Finally, it should be noted that nursing professionals should be fully involved in the process of recovery of the health of the people under their care, providing care based on scientific support, which will always strengthen the efficiency of the interventions carried out; that is why individualized care plans should be developed, useful in the training of future nursing professionals.

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