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Traumatic cerebrovascular injury

Lesión cerebrovascular asociada a la enfermedad traumática

Dear Editor,

Traumatic cerebrovascular injury (TCVI) is a rare complication of patients with traumatic disease with an incidence rate between 0.5% and 3.3%. Neurologic deficits—that can occur after the acute phase—happen in up to 58% of the patients with a mortality rate close to 25%.[1,2] It is important to identify risk groups so that optimal treatment can improve functional results and reduce the morbidity and mortality rates. We describe a series of cases approved by the local research ethics committee. The patients and/or their representatives' written informed consents were granted.

The identification of patients who can develop TCVI is one of the main challenges. Guidelines recommend using the modified Denver and Memphis criteria as screening.[3,4] High-energy mechanisms are the leading cause of TCVI, above all, those causing flexion-extension, rotation, and deceleration; anecdotically, low-energy trivial mechanisms like chiropractic, and the practice of yoga have been described.[5] In our series, all cases presented with closed trauma, being high-energy most of them, due to traffic accidents (60%). All showed traumatic brain injury. Clinical characteristics and severity scores are shown on Table 1.

The modified Denver and Memphis criteria include aspects associated with the lesion mechanism, associated lesions, and the clinical characteristics of patients with traumatic disease. However, despite such criteria, a non-negligible number of patients won't be diagnosed with TCVI.[1] In this sense, a recent study conducted by Leichtle et al.[6] estimates that up to 20% of the patients are misdiagnosed, and 25% of these have severity scores ≥ 3 according to the Denver scale; it is for this reason that authors recommend universal screening to discard TCVI in all patients with severe traumatic disease due to closed mechanisms. However, feasibility and cost-effectiveness studies are needed to back up this approach.

The severity of TCVI is defined based on the Denver severity scale (also called the Biffl scale), and has been designed to guide the therapeutic approach and for the prognosis of results.[4] However, this scale only defines lesions caused at arterial level at the TCVI normal setting sparing venous lesions on cerebral sinuses that can be accompanied by neurologic deficits.[5] We present 5 cases, 3 of which showed arterial lesions with severity grades II, III, and IV (Fig. 1A, B, E, F, and G) plus 2 cases of lesions in cerebral venous sinuses (Fig. 1C and D).

The therapeutic goal is to prevent the development of ischemic lesions.[3] Benefits in the morbidity and mortality rates have been demonstrated with the early use of antithrombotic therapy[4]; despite of that, there is discrepancy on what the most suitable antithrombotic therapy is (anticoagulation or antiplatelet therapy).[7] The risk of ischemic events increases the severity of the lesion; therefore, the current recommendations advocate for using antithrombotic therapy after diagnosis considering the bleeding risks involved.[3,4,8] In our series, 4 patients received anticoagulation with unfractionated heparin, and 1 with low-molecular weight heparin. One patient presented with bleeding (Fig. 1H) 5 days after starting anticoagulant therapy with unfractionated heparin. At the present time, no

Table 1 Demographic, clinical, and evolutionary characteristics of the cases.

Characteristics	Case #1	Case #2	Case #3	Case #4	Case #5
Age (years)	31	24	55	37	20
Sex	Feminine	Masculine	Masculine	Masculine	Feminine
Mechanism	Traffic	Traffic	Bicycle fall	Ordinary Fall (his own height)	Traffic
Modified Denver/Memphis criteria	1/1	2/2	0/0	1/1	3/1
Type of TCVI on the CAT	Dissection of right vertebral artery	Dissection of left supraclinoid ICA	Thrombosis of transverse venous sinus and jugular vein	Venous thrombosis of sigmoid sinus and jugular vein	Bilateral dissection of both ICAs
Denver scale (Biffl)	I (rosary-like pattern, good distal flow)	IV (filiform flow)	Non-applicable	Non-applicable	III (pseudoaneurysm)
RTS (scores)	7.84	7.84	6.9	7.84	4.3
GCS (scores)	13 (M6)	12 (M5)	9 (M5)	15 (M6)	4 (M1)
ISS/AIS head and neck (scores)	14/3	35/5	35/5	9/3	50/5
Cranial CAT	Epicranial hematoma (Marshall score I)	Epidural hematoma, SAH (Marshall score IV)	Hemorrhagic concussions, subdural hematoma, and venous epidural hemorrhage (Marshall score II)	Frontal concussion, SAH, subdural hematoma, and cerebellar epicranial hematoma (Marshall score II)	Ischemia in left MCA territory (frontoparietal and BG) (Marshall score III)
Anticoagulation	LMWH	UFH	UFH	UFH	UFH
Development of ischemic stroke	Yes. Infarction in the PICA territory	No	No	No	Yes. Infarction in the MCA territory
Admission-diagnosis time (days)	8	1	4	2	3
Type of neuromonitorization	Clinical/TDU	Clinical/TDU	ICP/$PTiO_2$	Clinical/TDU	ICP/$PTiO_2$
Days on IMV (days)	13	0	23	0	17
GOS after ICU/hospital discharge	3/3	3/5	2/4	4/5	3/3
ICU/hospital stay (days)	35/264	10/23	30/93	14/20	26/37

AIS, Abbreviated Injury Scale; BG, basal ganglia; CAT, computerized axial tomography scan; GCS, Glasgow Coma Scale; GOS, Glasgow Outcome Scale; ICA, internal carotid artery; ICP, intracranial pressure; ICU, intensive care unit; IMV, invasive mechanical ventilation; ISS: Injury Severity Score; LMWH, low-molecular weight heparin; M, motor; MCA, middle cerebral artery; PICA, posterior inferior cerebellar artery; $PTiO_2$, brain tissue oxygen pressure; RTS, Revised Trauma Score; SAH, subarachnoid hemorrhage; TCVI, traumatic cerebrovascular injury; TDU, transcranial Doppler ultrasound; UFH: unfractionated heparin.

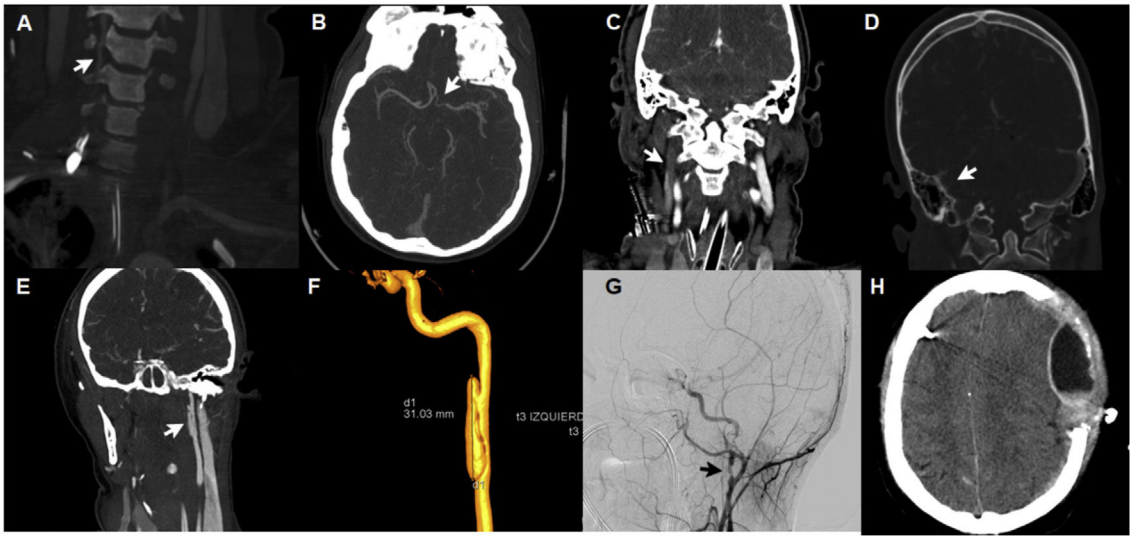

Figure 1 Radiographic aspects of patients with traumatic cerebrovascular lesion. **A:** dissection of right vertebral artery with rosary-like pattern and good distal flow (Denver type I). **B:** dissection of left supraclinoid internal carotid artery, filiform flow (Denver type IV). **C:** thrombosis of transverse venous sinus and right jugular vein. **D:** venous thrombosis of right sigmoid sinus. **E:** dissection of left carotid artery with development of pseudoaneurysm (Denver type III). **F:** arteriography with 3D reconstruction of pseudoaneurysm (3 cm of longitudinal axis) in the left carotid artery postbulbar extracranial segment. **G:** dissection of postbulbar extracranial segment with presence of 2 pseudoaneurysms in the right internal carotid artery. **H:** hyperacute epidural hematoma after starting anticoagulation therapy.

clinical trials have been conducted to guide the early use of antithrombotic therapy. Compared between the two, no therapy has been associated with a lower rate of ischemic lesions although most are low-quality studies with a high risk of bias.[7] Despite all this, unfractionated heparin is often advised for its reversibility, which is essential in the acute phase in addition to modified antiplatelet therapy.[3,7,9] Regarding patients with established ischemic lesions, antithrombotic therapy is not clear either. There is no evidence that dual antiplatelet therapy is more effective compared to the single-drug regime.[3]

Endovascular procedures are not considered a routine practice in low-grade lesions (I or II). However, they should be considered in more severe cases (grades III, IV, and V).[3,4] Low-grade lesions (I, and II) often have favorable progression; grade I lesions heal in 75% of the cases while grade II lesions will only do so in 8% of the cases, and 30% will progress into grade I lesions. Grade I lesions progress in 8% while grade II lesions do so in 40% of the cases.[5] High-grade lesions (III, IV, and V) often have worse progression; grade III lesions heal or improve 11% of the times but get worse in 25% of the cases. Grade IV lesions recanalize in 40% of the cases, but most of them show no changes at all.[5,10] In any case, a more severe lesion, complete occlusion or pseudoaneurysm should anticipate the use of the endovascular or surgical approach.

Overall, radiographic follow-up 7–10 days after diagnosis is advised; in case of vascular lesion resolution, the antithrombotic therapy could be suspended.[3] On the other hand, with persistent lesions, keeping antithrombotic therapy and performing a new imaging modality study at 6 months would be advised to reassess the need for moving on with therapy.[3,4]

In our series, the ICU stay was, on average, 26 days (IQR, 14–30) while the hospital stay was, on average, 37 days (IQR, 23–93). No patient died at the ICU or hospital discharge. Regarding functional results, ischemic lesion occurred in 2 patients.

TCVI is a preventable cause for stroke; its rapid detection and proper treatment are just essential to reduce the morbidity and mortality rates associated with TCVI. We believe it is of paramount importance that neurotrauma units develop protocols for the diagnosis and management of this condition.

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Mechanical power greater than 17 joules/min in patients with respiratory failure secondary to SARS-CoV-2 infection

Poder mecánico mayor de 17 julios/min en pacientes con insuficiencia respiratoria secundaria a infección por SARS-CoV-2

Dear Editor:

In mechanical ventilation (MV), the configuration of ventilation parameters is key for pulmonary protection purposes. The term mechanical power is used to define the amount of energy transmitted from the ventilator to pulmonary parenchyma in each respiratory cycle.[1] The most recent medical literature available confirms that in patients on MV due to SARS-CoV-2-induced respiratory failure, a MP threshold of 17 J/min could be associated with a higher risk of death.[2]

In this context, our objective was to study the association of MP with short-term survival (28 days) of patients admitted to an intensive care unit (ICU) due to SARS-CoV-2. Therefore, using data from our registry of patients with COVID-19 developed after gaining approval from the local research ethics committee and obtaining consent from the patients or their legal representatives (written or over the phone) we conducted a retrospective analysis of all the cases admitted to our ICU from March 2020 through July 2021 who had been mechanically ventilated due to SARS-CoV-2-induced ARDS. Sample was consecutive and divided into 2 different cohorts based on the value of MP within the first 24 h after endotracheal intubation: MP $\leq$ 17 J/min and MP > 17 J/min. MP was measured using the simplified formula proposed by Gattinoni et al.[3]

A descriptive analysis of the sample was initially conducted. Afterwards, a 28-day survival analysis was conducted with the Kaplan-Meier method for the variable of time of death (Log-rank test). To avoid confounding factors, a multivariable analysis of survival was conducted adjusting a Cox model (method: *forward;* introduction of variables to the model if $P < .2$, and exclusion of variables if $P > .5$; status: death at the ICU at 28 days; covariables used: age, the PaO2/FiO2 ratio prior to intubation, and the value of pulmonary compliance). Statistical significance was established at $P < .05$ for all analyses.

Out of the 565 patients admitted to the ICU with COVID-19 during the study period, only those with confirmed SARS-CoV-2 infections who received controlled MV were eventually analyzed. Also, the variables necessary to estimate MP in the supine position were estimated, after sedation and muscular paralysis, and within the first 24 h after starting MV.

Table 1 shows the main differences among the 79 patients analyzed categorized based on their MP.

The median survival times of both cohorts were 16 days (p25–75: 3–27) for the cohort of patients with MP $\leq$ 17 J/min vs 11 days (p25–75: 2–18) for the cohort of patients with MP > 17 J/min ($P = .02$). The group of patients with MP values > 17 J/min was associated significantly with greater chances of death at 28 days (OR, 2.91; 95%CI, 1.04–8.09; $P = .04$) (Fig. 1).

In the Cox regression analysis, values > 17 J/min of MP within the first 24 h after starting MV were independently