Medicina Intensiva 47 (2023) 411–415

medicina intensiva

http://www.medintensiva.org/en/

SCIENTIFIC LETTER

Vascular injury of the supra-aortic trunks in patients with traumatic brain injury

Lesión vascular de los troncos supraaórticos en pacientes con traumatismo craneoencefálico

Although infrequent, traumatic dissection of the internal carotid and vertebral arteries over their cervical trajectory is one of the leading causes of ischemic cerebral infarction (ICI) in young individuals. In this age group, the condition usually manifests in the absence of known risk factors, in the context of sports activities or secondary to cervical trauma, and the clinical manifestations (headache or neck pain, Horner's syndrome, neurological deficits, etc.), while known, do not always manifest as such. In this context, the diagnosis is often not established or is established late – with the resulting ominous consequences for the patient.[1] Ischemic cerebral infarction and its sequelae fundamentally result from the false lumen produced by endothelial or middle layer tearing, giving rise to stenosis, occlusion, pseudoaneurysms or embolization of the middle cerebral artery (MCA) from the thrombus formed at the dissection site.[2,3] The concurrence of supra-aortic trunk dissection (SATD) with severe or moderate traumatic brain injury (S-MTBI) has received little attention in the medical literature, due to the diagnostic difficulties involved (the clinical manifestations tending to overlap or be confused) or to the inherent seriousness of traumatic brain injury (TBI), demanding immediate intervention. In this context, diagnostic omission or delay is even more common.[4,5] Although the incidence of supra-aortic trunk dissection in the context of S-MTBI has not been clearly established, some publications estimate an incidence of 1%–9%, with a mortality rate of 20%–30%.[6,7] The early diagnosis of SATD is crucial, since surgical or endovascular treatment (thrombectomy or stent placement) and anticoagulation or antiplatelet therapy,[4] improve the prognosis. Digital subtraction angiography is the diagnostic technique of choice,[8] though other tools such as CT angiography or MRI angiography are initially more often used in situations where dissection is suspected. The use of transcranial Doppler ultrasound (TCD) in the management of S-MTBI has become consolidated in Neurocritical Care Units, given the hemodynamic information it provides through calculation of the flow velocity (FV) in the arteries of the circle of Willis.[5] However, very little has been mentioned on the screening capacity of TCD in detecting possible SATD in S-MTBI, even in recent publications,[9] though some authors have already demonstrated that asymmetries of the FV and pulsatility index (PI) between both MCAs are valid suspicion criteria.[10] We present a series of 15 patients with S-MTBI and SATD out of a total of 213 individuals with S-MTBI admitted to the Neurocritical Care Unit during the period 2017–2019, of which 50 had died at 6 months, underscoring the potential screening usefulness of TCD.

The main patient data are reported in Tables 1 and 2. In our series, all the patients were males with a mean age of 34 ± 13 years, and with no risk factors except for one case of Ehlers-Danlos syndrome. Traffic accidents predominated. Based on the Glasgow Coma Score (GCS), 11 patients were classified as presenting severe TBI (GCS ≤ 8 points) and four as presenting moderate TBI (GCS ≤ 14 points), with a predominance of diffuse lesions in the CAT exploration. Dissection mainly affected the internal carotid arteries, and in only two cases did dissection affect the vertebral arteries alone. Except for case 13, which manifested with Horner's syndrome, the clinical manifestations in the rest of the cases were indistinguishable from those possibly attributable to TBI. The TCD study evidenced asymmetry, expressed as a mean 22% decrease in the FV and PI of the MCA corresponding to the damaged homolateral vessel, in all but two cases – one due to patient circulatory arrest. With the exception of case 4, where suspicion was delayed for 13 days, the mean time from suspicion to the diagnosis of dissection was 10 h. Computed tomography angiography was the technique most often used to establish the diagnosis, while anticoagulation or antiplatelet therapy was the most commonly used treatment – only 5 cases being subjected to stent placement. As a consequence of dissection (Table 2), 11 patients suffered cerebral infarction, fundamentally in the territory of the MCA. The exception was case 1, where the territory corresponding to the occluded vessel of the posterior circulation was affected. The mortality rate at 6 months was 20% (3 cases). According to the modified Rankin score, at 6 months only four patients had a good outcome; three suffered moderate disability; and 5 patients suffered a severe disability.

Despite the few patients involved, our study describes one of the most extensive and homogeneous series in this field, since the majority of publications on SATD are set within the context of polytraumatisms or trauma limited to the cervical region. In coincidence with the observations of other authors, in our sample the incidence of SATD was low, associated with high-energy TBI, and with similar mortality figures fundamentally related to the severity of TBI. In contrast, the important disability was associated with the

DOI of original article: https://doi.org/10.1016/j.medin.2023.02.009

Table 1 Characteristics of the studied series.

Case	Age	RF	Cause of TBI	TCDB	Vascular injury	Initial clinical manifestations
1	36	No	Motorcycle	6	RCA + PICA	No focality
2	51	No	Motorcycle	2	RICA	Left hemiparesis
3	15	E. Danlos	Swimming pool	1	LICA+	Right hemiparesis
4	34	No	Motorcycle	6	LICA	Right hemiparesis
5	29	No	Sports	1	LICA	No focality
6	28	No	Fall	2	RCA	Profound coma
7	15	No	Fall	6	RICA	Profound coma
8	42	No	Run over	2	LICA	No focality
9	23	No	Car collision	2	LICA	Right hemiparesis + aphasia
10	57	No	Fall from height	6	RICA	Left hemiparesis
11	29	No	Motorcycle	1	RICA	No focality
12	50	No	Motorcycle	1	RICA + LICA	Right + left hemiparesis
13	45	No	Motorcycle	2	LICA + RCA	Horner + 3rd cranial nerve paralysis
14	46	No	Motorcycle	6	LICA	Right hemiparesis
15	47	No	Car collision	5	RICA	Left hemiparesis

RF: Risk factor; TBI: Traumatic brain injury; TCDB: Lesion type according to Traumatic Coma Data Bank; RCA: Right cerebral artery; PICA: Posteroinferior cerebellar artery; RICA: Right internal carotid artery; LICA: Left internal carotid artery; E. Danlos: Ehlers Danlos syndrome; Horner: Horner's syndrome.

Table 2 Diagnostic tests,treatment and evolution of patients.

Case	TCD MCA	Time from suspicion to diagnosis	Definitive diagnosis	Treatment	Dissection brain injury	GOSE at 6 months	mRankin at 6 months
1	Asymmetry	48 h	CTangio	Antiplatelet	Infarction	3	4
2	Asymmetry	2 h	CTangio	Antiplatelet	Infarction	5	1
3	Asymmetry	24 h	DigitAngio	Stent	Infarction	5	3
4	Asymmetry	13 days	DigitAngio	Stent	Infarction	5	4
5	Asymmetry	20 min	CTangio	Antiplatelet	No	8	0
6	Asymmetry	1 h	CTangio	Anticoagulation	No	3	4
7	No	30 min	CTangio	No	Infarction	1	Death
8	Circulatory arrest	15 min	CTangio	Anticoagulation	No	8	1
9	Asymmetry	20 h	CTangio	Anticoagulation	Infarction	4	3
10	Asymmetry	10 h	CTangio	Stent	Infarction	1	Death
11	Asymmetry	30 min	DigitAngio	Antiplatelet	No	8	0
12	Asymmetry	7 h	CTangio	Stent	Infarction	3	3
13	Asymmetry	40 min	CTangio	Anticoagulation	Infarction	2	5
14	Asymmetry	15 min	CTangio	Antiplatelet	Infarction	3	4
15	Asymmetry	24 h	DigitAngio	Stent	Infarction	1	Death

CTangio: computed tomography angiography; DigitAngio: digital subtraction angiography; Antiplatelet: antiplatelet medication. Anticoagulation: anticoagulant therapy; GOSE: Extended Glasgow Outcome Scale; mRankin: modified Rankin score 8290 min (187,209) 138 h mean 10 h.

extent of the cerebral infarction caused by the vascular injury. Another observation of note is the usefulness of TCD for suspecting SATD in the context of TBI. Although a difference in the FV values between the cerebral hemispheres may be explained by the presence of space-occupying lesions or greater inflammation in one hemisphere, in this case the decrease in velocities was accompanied by an increase in PI secondary to the elevation of intracranial pressure. In contrast, a decrease in FV associated with a drop in PI is suggestive of an ischemic process, where the decrease in PI expresses the attempt to compensate the perfusion defect with a decrease in distal cerebrovascular resistance or through collateral circulation. Possibly, the routine use of TCD in our study can explain why the time elapsed from suspicion to the diagnosis of SATD was shorter than in other publications − with the exception of the single case characterized by an excessive diagnostic delay.

We are of the opinion that in all patients with S-MTBI, the possibility of SATD should be considered and either confirmed or discarded as soon as possible. Although CT angiography is the most accessible and reliable screening tool, the findings may sometimes prove negative if the technique is performed very early. Transcranial Doppler ultrasound, which can be repeated as often as needed, may be an option capable of securing earlier detection.[1,2,10]

References

1. Rommel O, Niedeggen A, Tegenthoff M, Kiwitt P, Bötel U, Malin J. Artery injury following severe head or cervical spine trauma. Cerebrovasc Dis. 1999;9:202–9.
2. Srinivasan J, Newell DW, Sturzenegger M, Mayberg MR, Winn HR. Transcranial Doppler in the evaluation of internal carotid artery dissection. Stroke. 1996;27:1226–30.
3. Wang GM, Xue H, Guo ZJ, Yu JL. Cerebral infarct secondary to traumatic internal carotid artery dissection. World J Clin Cases. 2020;8:4773–849.
4. Kowalski RG, Haarbauer-Krupa JK, Bell JM, Corrigan JD, Hammond FM, Torbey MT, et al. Acute ischemic stroke after moderate to severe traumatic brain injury: incidence and impact on outcome. Stroke. 2017;48:1802–9.
5. Aries MJ, De Jong BM, Uyttenboogaart M, Regtien JG, van der Naalt J. Traumatic cervical artery dissection in head injury: the value of follow-up brain imaging. Clin Neurol Neurosurg. 2010;112:691–4.
6. Paiva WS, Morais BA, de Andrade AF, Teixeira MJ. Mild traumatic brain injury associated with internal carotid artery dissection and pseudoaneurysm. J Emerg Trauma Shock. 2018;11:151.
7. Esnault P, Cardinale M, Boret H, D'Aranda E, Montcriol A, Bordes J, et al. Blunt cerebrovascular injuries in severe traumatic brain injury: incidence, risk factors, and evolution. J Neurosurg. 2017;127:16–22.
8. Miller PR, Fabian TC, Croce MA, Cagiannos C, Williams JS, Vang M, et al. Prospective screening for blunt cerebrovascular injuries: analysis of diagnostic modalities and outcomes. Ann Surg. 2002;236:386–93.
9. Robba C, Taccone FS. How I use Transcranial Doppler. Crit Care. 2019;23:420.
10. Bouzat P, Francony G, Brun J, Lavagne P, Picard J, Broux C, et al. Detecting traumatic internal carotid artery dissection using transcranial Doppler in head-injured patients. Intensive Care Med. 2010;36:1514–20.

Ana María Ferrete-Araujo[a,b,*], Daniel A. Godoy[c,d], Francisco Murillo-Cabezas[b]

[a] *Unidad de Neurocríticos, Servicio de Medicina Intensiva, Hospital Universitario Virgen del Rocío, Seville, Spain*
[b] *Departamento de Medicina, Facultad de Medicina, Instituto de Biomedicina de Sevilla (IBiS)/Centro de Investigaciones Científicas (CSIC)/Universidad de Sevilla, Seville, Spain*
[c] *Unidad de Cuidados Neurointensivos, Sanatorio Pasteur, San Fernando del Valle de Catamarca, Catamarca, Argentina*
[d] *Unidad de Terapia Intensiva, Hospital San Juan Bautista, San Fernando del Valle de Catamarca, Catamarca, Argentina*

[*] Corresponding author.
E-mail address: amferretearaujo@gmail.com
(A.M. Ferrete-Araujo).

Validation of the P/FPE index in a cohort of patients with ARDS due to SARS-CoV-2

Validación del índice P/FPe en una cohorte de enfermos con SDRA secundario a SARS-CoV-2

Based on the limitations that the partial pressure arterial oxygen (PaO_2)-fraction of inspired oxygen (FiO_2) ratio shows in the classification of severity in patients with acute respiratory distress syndrome (ARDS),[1] recently, Martos-Benítez et al.[2] assessed the severity of ARDS using the FiO_2-adjusted PaO_2 and positive end-expiratory pressure (PEEP): PaO2/(FiO_2 x PEEP) or P/FPE ratio.[3] Due to the differences between ''traditional'' ARDS and ARDS due to COVID-19 (C-ARDS) like the underlying cause, mechanism of pulmonary lesion, clinical presentation, and therapeutic management,[4] we believe that the hypothesis that the utility of the P/FPE ratio to predict mortality in C-ARDS is not the same for patients with ''traditional'' ARDS.

DOI of refers to article: https://doi.org/110.1016/j.medin.2023.03.008

A cohort of 507 patients with C-ARDS treated at Hospital Universitario Marqués de Valdecilla, Santander, Spain, and another cohort of 217 patients with ''traditional'' ARDS previously published by Martos-Benítez et al.[2] were studied using the inclusion and exclusion criteria described by them. In both cases, the registries used had been accepted by the local Research Ethics Committee of each center. In the Spanish series, informed consent from the patients/legal representatives (written or over the phone) was required. In the Cuban series consent was not required due to the retroactive nature of the data collected. The severity of ARDS was evaluated within the first 24 h after starting invasive mechanical ventilation with the patient while in the supine position. All individuals were categorized as having mild, moderate or severe ARDS according to the Berlin definition.[5] Based on the P/FPE ratio, patients were categorized into mild ($40 < P/FPE\ ratio \leq 60$), moderate ($20 < P/FPE\ ratio \leq 40$) or severe ARDS ($P/FPE\ ratio \leq 20$).

Descriptive statistics and a multivariate logistics regression analysis to explore the impact of ventilatory configurations and respiratory indices on in-hospital mortality were used. The area under the operator receiving characteristics curve (AUC) was used to assess the performance of respiratory indices. A multivariate logistics regression analysis was used to explore the impact of respiratory indices with the following primary endpoint: 28-day mortality rate. To inter-