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Archives of Emergency Medicine and Critical Care

Case Report

Interest of Transcranial Doppler in the Follow-Up of the Brain-Damaged Patient: About Two Cases

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Submitted: 03 January 2024
Accepted: 13 February 2024
Published: 16 February 2024

ISSN: 2476-2016

Keywords
- Echo-Doppler Transcranial Doppler (EDTC)
- Brain damaged
- Infusion pressure cerebral (PPC)
- Cérébral blood flow (CBF)

Abstract

Monitoring brain-injured patients using transcranial Doppler ultrasound (TCDUS) has profoundly changed our therapeutic attitude. Indeed, EDTC is a non-invasive technique that can be performed at the patient's bedside. It not only allows us to detect any changes in cerebral perfusion pressure(CPP) early by monitoring cerebral blood flow (CBF), but also to suspect a state of brain death.

We report here the case of the treatment of two brain-injured patients, one of whom was post-road accident (AVP) with a fatal outcome and the other with a hemorrhagic type stroke with a favourable outcome, both tracked using the EDTC.

INTRODUCTION

Transcranial Doppler Ultrasound (TCED) is an ultrasound technique that has been described for several decades, whose objective is to evaluate the velocities of red blood cells within the cerebral arteries and particularly the middle cerebral artery (MCA) using ultrasound. This represents 60% of homolateral cerebral blood flow [1]. It is a means of studying cerebral hemodynamics at the patient's bedside [2]. It provides information on cerebral blood flow (CBF) which results from cerebral perfusion pressure resulting from the difference between mean arterial pressure (MAP) and intracranial pressure (ICP) (PPC=MAP-PIC) [3]. Its reproducibility and speed of implementation have made it a technique of choice in neurotraumatology where the time factor is crucial for the prognosis [4]. The brain injured by trauma is a common cause of admission to the intensive care unit. If the severity of the initial brain lesions is assessed by the clinic and computed tomography (CT), these are poorly predictive of their evolution [5]. The EDTC would therefore be used to assess this risk, as well as the evolution in real time by measuring the cerebral perfusion flow and therefore optimize the treatment.

CLINICAL CASE 1

Mr EBS, 24 years old, énolo-tabacco addict, was admitted to the intensive care unit at the Military Training Hospital of Akanda (HIAA) for the treatment of a severe head injury following a road traffic accident (AVP) on a car-pedestrian mechanism (direct left lateral impact with projection onto the asphalt).

On admission to the emergency department (SAU), within an hour of the accident, the patient was found to be in an altered state of consciousness (Glasgow score at 7/15), with generalised tonic-clonic convulsions, reactive isochore pupils, hemodynamic stability (PAM at 92 mmHg), a Superficial polypnea (22 cycles/min), and a low abundance otorragia on the left.

After conditioning in the emergency department with oxygen therapy using a simple facial mask, cervical immobilization using a rigid collar and placement of a peripheral venous lane (VVP) of good caliber, he underwent a whole body TDM which revealed a Fisher 2 meningeal haemorrhagia in the cephalic region,, with no extra or sub-dural haematoma or midline deviation , and a

pneumoencephaly on the left with a fracture of the rock on the left (Figure 1), on the thoracic region, we found minimal bilateral pulmonary contusions.

On the biological side, apart from a moderate liver cytolysis and a lowered TP to 57%, the rest of the haematological and metabolic assessment is without particularities.

The patient was immediately transferred to the intensive care unit, where he benefits from an EDTC that returned normal (Figure 2), before being intubated, ventilated and sedated on the basis of the neurological and respiratory criteria. He also benefits from the implementation of a central venous path in the left internal jugular under guided echo and a gastric probe.

The rest of the treatment consists in preventing and fighting against secondary cerebral attack of systemic origin (ACSOS) and/or central origin (ASCOC) by the mean of Levetiracetam in addition to Midazolam in the PSE (prevention of convulsive crises). Other therapeutic traitements, included anti-tetanus serotherapy and vaccination, prevention of meningeal infection with ceftriaxone (100mg/kg/24h) and vitamin B1 and B6 supplementation.

Thirteen hours after his admission to intensive care unit, the patient has a brutal pupillary modification in the form of left anisocoria. The EDTC made immediately revealed a high pulsatility index (IP) at 1.42 with a diastolic diameter of the ACM to 20.52 cm/s (Figure 3), leading to the conclusion of HTIC. The second cerebral CT done in emergency shows a worsening of brain damage compared to admission. 10% mannitol osmotherapy (at the rate of 1g/kg/24h) is started and the control EDTC shows an improvement in the HTIC with an IP rising to 0.94 (Figure 4), motivating the stop Osmotherapy.

After several checks of EDTC checks that returned normal, the patient benefits from the cessation of sedation on the 6th day of hospitalization. However, on the 7th day the onset of an intermediate mydriasis as well as an IP at 1.67, VD 7.83 cm/s and vs 33.11 cm/s (Figure 5), were correlated with dysnatremia motivating its correction and resumption of sedation.

The appearance on the 8th day of hospitalization of a reactive bilateral mydriasis with the absence of reflex of the brainstem and EDTC showing retrograde diastolic flow with virtually no diameter of the ACM in diastole, led to the suspicion of brain death (Figure 6). The CT scan carried out 3 hours earlier (Figure 7), showed the intra-parenchymal haematomes in the left parieto-occipital and right fronto-temporal areas surrounded by perilesional oedema (12 x 10 mm) without anomaly of the midline and ventricular system. The patient died less than 12 hours later on D8 of hospitalization.

CLINICAL CASE 2

Mr B.B, 38 years old, with known neglected hypertension, was admitted to the intensive care unit of the Akanda Armies Training Hospital (HIAA) for the management of a hemorrhagic stroke, classified as ICH 2 (Figure 8). On admission to the intensive care unit, the patient presented an obnubilation with GCS at 14/15, a psychomotor agitation and a HTA at Grade II. The biological assessment was without abnormality. His management consisted on preventing secondary cerebral attacks of systemic origin (ACSOS) and/or central origin (ASCOC), balancing blood pressure with nicardipine in the PSE, preventing convulsions with Valproic acid at 1.5g/24 hours, preventing complications of alcohol withdrawal by administration of vitamin B1 and B6. We note that before the patient's agitation state, he benefited empirically from Mannitol on D1.

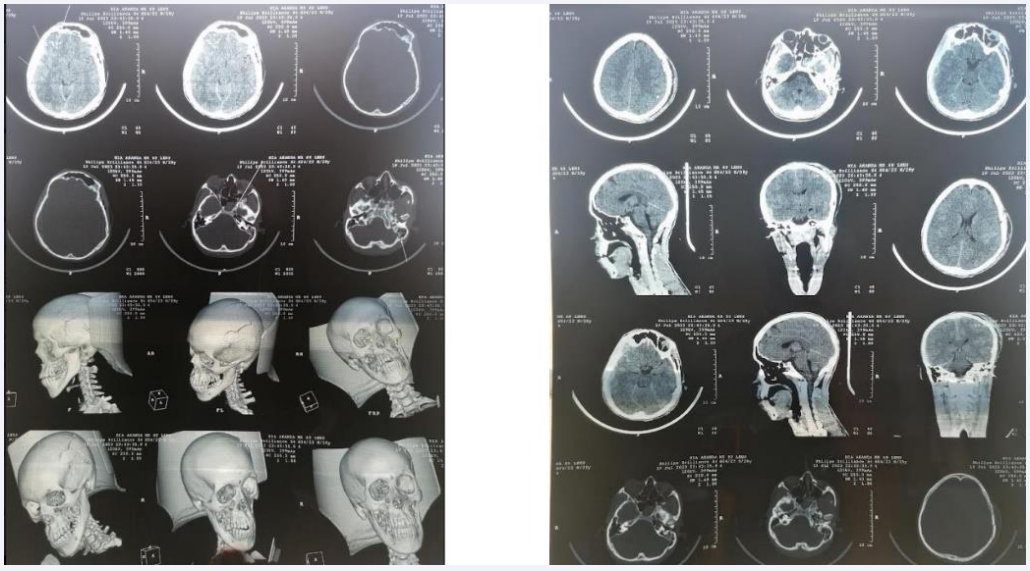

Figure 1 CT scan at admission

Figure 2 EDTC at H1 on admission

Figure 3 EDTC at H1

Figure 4 EDTC at H30

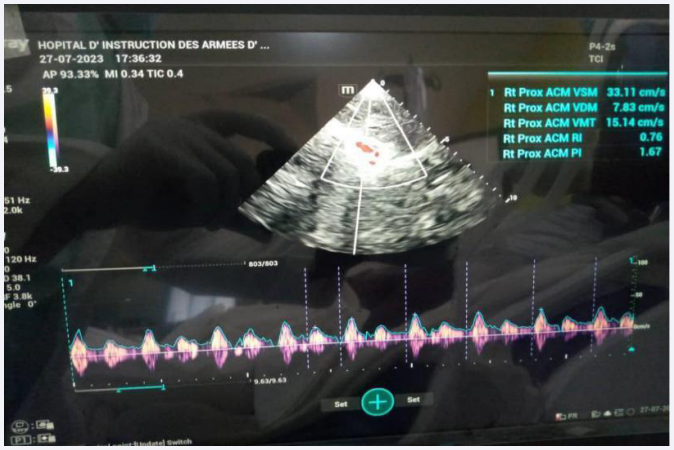

Figure 5 EDTC at J7

Figure 6 EDTC at J8 (brain death)

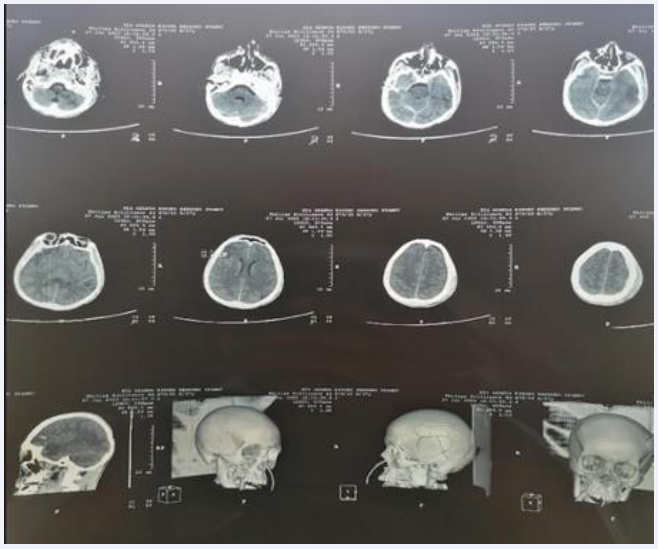

Figure 7 CT scan at J7 follow up

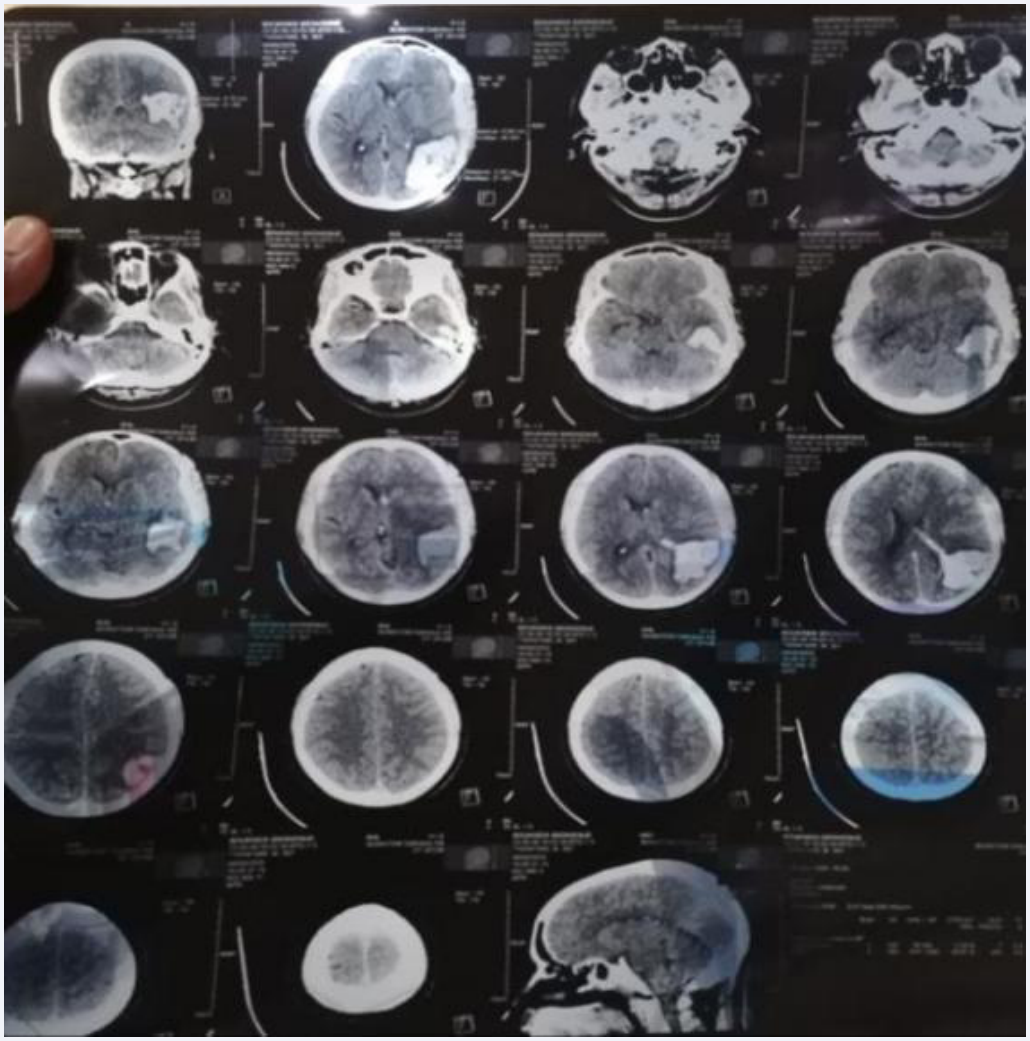

Figure 8 CT scan at AVCH patient 2.

The evolution under this treatment was marked by the persistence of a psychomotor agitation and mental confusion, motivating the realization of an EDTC, which revealed a decrease in PPC with a hight IP of 1.32, and lower Vd and Vs (photo 9) suggesting a cerebral hypoperfusion of brain origin secondary to HTIC for which he received osmotherapy. The control 2h after administration of the mannitol highlights a normal IP at 1.12, a decrease in Vd at 25.78 cm/s and the VS to 69.15 cm/s compatible with a hypoperfusion of systemic origin (photo 10) secondary to the 'Osmotherapy and motivating a filling. The evolution of the patient was favourably with resolution of the confusion and the psychomotor agitation, and he was discharged at D7 of hospitalization.

DISCUSSION

This clinical case shows the importance of using EDTC in monitoring and decision-making in case of head trauma and AVCH. It enables us to monitor changes of cerebral haemodynamics and to adapt our care strategy.

In fact, this clinical case shows that it was the repeated monitoring of cerebral haemodynamics that enable us to detect early the occurrence of cerebral hypoperfusion of cerebral origin (secondary to HTIC) or systemic origin.

EDTC allows the estimation of intracranial blood flows by assessing the velocity of red blood cells within the cerebral arteries, and particularly ACM [6]. The blood flow is measured in velocities (v = cm/s) and this velocity depends on two parameters: the ultrasound emission frequency (F) and the alpha angle formed by the vessel, which gives an indirect approach to the flow rate (Q) in the vessel. This flow is graphically represented with the time and the frequencies (speeds) in abscissa. The curve obtained enable the measure of systolic velocity (Vs), diastolic velocity (Vd) and average velocity (Vm) whose values are specific to each vessel studied (Table 1). The measurement of these velocities allows the calculation of the pulsatility index (IP)

Figure 9 EDTC at J3 (Patient AVCH)

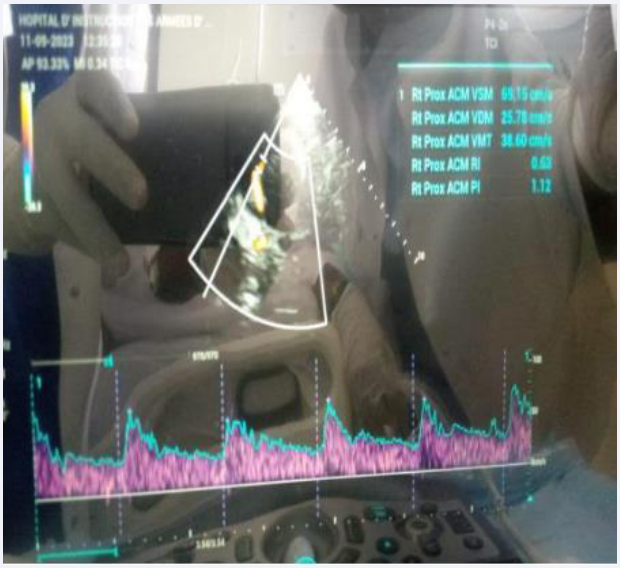

Figure 10 EDTC at H2 post mannitol

described by Gosling: IP = (Vs-Vd)/Vm, whose normal value in adults is 0.71 ± 0.10 (Table 1). All changes in cerebral resistances will result in a modification of the Vd and the IP (1). An increase of the intracranial pressure increases the downstream resistance and this results in a decrease in Vd and an increase of IP [7]. In our case, the diagnosis of HTIC was suspected by the worsening of the neurological state and the appearance of an aisochoria, but confirmed by the EDTC with an IP at 1.42 and Vd at 20, 52 compared with the IP at 1.17 and Vd at 49.05, on admission, allowing us to start osmothérapy with mannitol (1g/kg) in addition to mechanical ventilation. The duration of osmotherapy was determined by its effects on cerebral haemodynamics, visible to EDTC in less than 24 hours (IP at 0.94 and Vd at 31.34).

The EDTC therefore allowed us to adapt our management as shown in the study by Ract et al., in which patients with abnormal EDTC values on admission benefited from the same treatment to restore normal cerebral perfusion pressure [8].

After 7 days of hospitalization, a follow-up CT scan showed an improvement in the meningeal haemorrhage without a sign

Table 1: Normal values of the parameters measured at EDTC in adults [3]

Artery	Pathway	Deepness(cm)	Vm(cm/s)	Sense of flow
Average brain artery	Temporal	45-60	62±12	+
Anterior brain artery	Temporal	60-75	51±12	-
Posterior brain artery	Temporal	70-90	44±11	+
Carotid siphon	Occipital	50-75	42±10	+
Basilaire trunk	Under occipital	70-90	40±8	-

of franc HTIC despite the presence of diffuse cerebral oedema. It was the appearance of pupillary changes such as bilateral reactive mydriasis associated with the EDTC which objective IP at 1, 67 and VD at 7.83 evocating a cerebral hypoperfusion which motivates the resumption of osmotherapy and correction of the ACSOS (hyponatremia). After 16 hours, we notice at EDTC a disappearance of diastolic velocities and the presence of a pendulum infusion, which makes it possible to strongly suspect a state of brain death and to initiate a limitation of therapeutic acts.

The diagnosis of brain death is based on EEG and/or arteriography, but EDTC has very good sensitivity and satisfactory specificity [9-11]. Given the lack of available resources and the difficulty of access to the EEG in our sub -Saharan countries, the EDTC remains crucial to contribute to the diagnosis of brain death in addition to the loss of reflexes of the brainstem. EDTC in Gabon is advised because we have no clear legislation framing the diagnosis of brain death.

For the clinical case n ° 2 on the AVCH, the EDTC carried out at 2 hours from the 10% mannitol administration allowed us to highlight a normalization of the IP (therefore disappearance of HTIC) but also to identify early the fall of Vds and Vs compatible with systemic hypoperfusion and secondary to this same osmotherapy.

CONCLUSION

Traumatic brain injury is an evolutive disease, and the management is intended to maintain a sufficient cerebral blood flow. Several monitoring techniques exist, and several examinations help to follow the evolution of this pathology, but EDTC remains the most reproducible technique. It is quick to perform, making it possible to search as many times as necessary, the existence of HTIC or suspect brain death. It has become an essential tool in Intensive Care Unit and its ease of use as well as its safety have made it the choice in the management and monitoring of brain injury. Our various cases confirm the importance of its use in this type of patient, despite that an initial CT scan is not predictive of an unfavourable outcome. It should therefore be available and used in Intensive Care Unit for close monitoring of these cerebro-injured patients.

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