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Reliability of Computed Tomographic Angiography in the Diagnosis of Brain Death

J-C. Combes, A. Chomel, F. Ricolfi, P. d'Athis, and M. Freysz

ABSTRACT

Objective. This study examined the validity of cerebral computed tomographic (CT) angiography in the diagnosis of brain death (BD) compared with conventional cerebral angiography.

Methods. This prospective, monocentric study was performed over a 24-month period and included 43 patients, at least 18 years of age, with clinical criteria of BD. All patients underwent cerebral CT angiography and then cerebral angiography. To confirm BD, the CT scan had to show the absence of perfusion of A2 anterior cerebral artery segments (A2-ACA), M4 middle cerebral artery segments (M4-MCA), P2 posterior cerebral artery segments (P2-PCA), basilar artery, internal cerebral veins, and finally the great cerebral vein. Cerebral angiography showed cerebral blood flow arrest at the level of the foramen magnum for posterior circulation and carotid siphon for anterior circulation.

Results. For 30 patients, BD was confirmed by both examinations. For 13 patients, cerebral angiography confirmed BD, whereas CT angiography still showed cerebral perfusion; the divergence rate was 30.2%.

Conclusions. CT angiography seems to be a promising exam to confirm BD. However, the divergence with cerebral angiography is significant mainly concerning A2-ACA, which are proximal. It may be possible to only use the absence of opacification of M4-MCA, P2-PCA, basilar artery, and venous blood return to remain in conformity with the French law. In all cases, the international medical community should obtain a consensus for the interpretation of CT angiography to use it extensively as a complementary exam for BD.

BRAIN DEATH (BD) is the consequence of cerebral blood flow arrest leading to total and irreversible loss of hemispheric and brain stem functions.[1] In France, BD criteria are precisely defined by decree[2]: complete and persistent absence of consciousness and spontaneous movement, no brain stem reflexes, and no spontaneous breathing on apnea challenge despite hypercarbia.[3] In some countries, this clinical diagnosis is sufficient. Nevertheless, in others and in particular in France, confirmatory examinations are required[4]: either electroencephalography (EEG) or cerebral angiography.[2,5,6]

EEG must be performed under well-defined conditions[2,7]: several potentially reversible conditions, such as drug intoxication or hypothermia must be excluded. Furthermore, EEG does not assess brain stem activity. It imposes a delay for the diagnosis because 2 separate EEGs are necessary, and it is only available during working hours. Despite these limits,[8,9] it is the most commonly used test to confirm the clinical signs of BD because it is easily performed at the patient's bedside.

Conventional cerebral angiography must show intracerebral circulatory arrest at the entry to the skull.[5] Among radiological examinations, it remains the gold standard to confirm the diagnosis of BD. It may also be useful each time clinical examination or EEG is compromised by the use of barbiturates, for instance. It can be utilized only if the hemodynamic status is adequate: it requires patient trans-

From the Departments of Anesthesiology and Intensive Care (J.-C.C., A.C., M.F.) and Radiology (F.R.), Dijon University Hospital, General Hospital, and the Department of Biostatistics, Dijon University Hospital, Bocage Hospital (P.A.), Dijon, France.

Address reprint requests to Anne Chomel, MD, Department of Anesthesiology and Intensive Care Unit, CHU Dijon University Hospital, 3 rue du Faubourg Raines, 21033 Dijon, Cedex, France.

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portation to an angiography suite. The exam is performed by injection into 4 vessels (2 common carotid and vertebral arteries). Thus, it is an invasive examination which needs an experienced neuroradiologist and the availability of an angiography suite. Digital subtraction angiography performed by venous infusion is easier and less invasive but also requires an angiography suite.[10,11]

The use of a computed tomographic (CT) scan to diagnose BD was proposed as early as 1978,[12,13] then again in 1985 as an alternative to cerebral angiography.[14] More recently, spiral CT angiography was described as an angiographic technique by venous infusion.[15] This exam developed widely these last years thanks to a new generation of multirow CT which allows visualization of opacified cerebral vessels. Some criteria for validity have been proposed.[16] The purpose of this work was to evaluate CT angiography compared with cerebral angiography to diagnose BD and to suggest radiological criteria for this diagnosis.

MATERIALS AND METHODS

This prospective, monocentric study, performed over a 24-month period, was approved by our local ethics committee, and included all patients at least 18 years of age with suspicion of BD according to clinical criteria defined by law[2]: complete and persistent absence of consciousness and spontaneous movement, no brain stem reflexes, and no spontaneous breathing on apnea challenge despite hypercarbia. There were no exclusion criteria. After the closest relative agreement, CT scan was performed without and with injection of contrast material, followed by cerebral angiography. During these exams, mean arterial blood pressure was maintained above 65 mm Hg.

Concerning CT scan (GE High Speed), there were 3 spiral CT scan phases. The first one (which was the reference) was performed without contrast medium, in order to show cerebral parenchyma. The second one was realized 25 seconds after the start of contrast medium injection, and the third one 60 seconds after the beginning of the second phase. Images were acquired at a slice thickness of 7 mm with pitch of 1. The brain was imaged with the following parameters: kV, 120; mA, 130; duration, 30 seconds; entry angle, parallel to orbitomeatal line; range, C1-2 cervical level to vertex; FOV, 230 mm; Matrix, 512^2. Images were reconstructed with 3.5 mm increments. The injection was performed with nonionic contrast material (100 mL of iopromide 370, concentration above 300 mg/mL) into a basilic or cephalic vein of the arm, at a rate of 4 mL/s using a power injector and an 16-gauge catheter. The opacification of the superficial temporal arteries on the second helical scan attested that the injection was performed under adequate hemodynamic conditions. On the third helical scan, the criteria diagnosing cerebral circulatory arrest were the nonopacification of the A2 segment of the right and left ACA, M4 segment of the right and left MCA, P2 segment of the right and left PCA, basilar artery, right and left internal cerebral veins, and finally the great cerebral vein. All the criteria were necessary to establish BD.

Cerebral angiography (Philips biplan alura) was performed immediately after CT angiography. The injection was performed with nonionic contrast material (60 mL of iopromide 370, concentration above 300 mg/mL), at a rate of 15 mL/s using a power injector and the same vein as that for the spiral CT scan. Imaging was performed in the lateral projection. The acquisition delay of images was 5 seconds. The last image was realized at least 60 seconds after the injection. The diagnosis of BD was confirmed by visualization of cerebral blood flow arrest at the level of the foramen magnum for the posterior circulation and the carotid siphon for the anterior circulation.

These 2 examinations were interpreted by 2 radiologists trained in CT angiography and conventional angiography. The radiologist who interpreted CT angiography was unaware of the results of cerebral angiography.

The data were evaluated statistically: as all patients were brain dead according to conventional angiography, the frequency of diagnosis confirmed by CT angiography is the sensitivity of this examination, but the specificity could not be estimated. The agreement between examinations was evaluated without the Kappa index, which only gives a trend of concordance, but with a 95% confidence interval (CI), using a binomial distribution, for the theoretical frequency of divergence between the 2 techniques. The characteristics were compared using the chi-square test for qualitative data and the Mann-Whitney test for quantitative data. A value of $P \leq .05$ was considered significant. The results are presented as mean values ± SD (herein SD values are presented in parentheses).

RESULTS

We screened 43 patients of mean age 50.2 (15.2) years and sex ratio (men to women) of 3.77. The diseases were severe head trauma (48.8%), stroke (23.3%), cerebral aneurysm rupture (23.3%), meningitis (1 patient), and cerebral anoxia by cardiac arrest (1 patient).

Their mean blood pressure was 97.4 (22.6) mm Hg during CT angiography. The delay between the request and the CT angiography was 3.27 (3.81) hours and the mean duration of the exam was 16.8 (9) minutes. The mean duration of cerebral angiography was 12.6 (6.6) minutes. The delay between clinical BD and CT angiography was 9.29 (7.57) hours. At the moment of CT angiography, EEG was available for 21 patients only.

The sensitivity for CT angiography was 69.7%. For 30 patients (group 1), the diagnosis of BD was confirmed by both exams and for 13 patients (group 2), CT angiography suggested cerebral opacification, whereas cerebral angiography established BD: this corresponded among the 43 patients to a divergence frequency of 30.2% (95% CI range from 17.8–46.1%). Data from these 2 groups are detailed in Table 1: they do not differ statistically for age, sex ratio, hemodynamic status, and BD etiologies. The delay to realize CT angiography was longer for the second group, but the difference was not statistically significant. Also, the difference is neither significant for the delay between clinical BD and CT angiography, nor for the delay between the end of CT angiography and the beginning of cerebral angiography.

In the study, only 1 patient (patient 32) showed a deep venous blood return on the CT scan, at the level of the great cerebral vein. The same patient had opacification of A2-ACA, whereas cerebral angiography, realized 20 minutes after CT angiography, confirmed BD. No patient showed perfusion in the vertebrobasilar territory and in P2-PCA.