Scintigraphic Evaluation of Brain Death: Significance of Sagittal Sinus Visualization

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Radiotracer scintigraphy has been commonly used in this country to confirm and document the clinical diagnosis of brain death. Whether the presence of radiotracer activity in the region of sagittal venous sinus (SVS) represents actual blood flow to the brain in the absence of demonstrable cerebral arterial flow remains a controversial issue. Our retrospective study was performed to review the significance of such sagittal tracer activity. Of the 53 patients showing no cerebral arterial flow, 26 showed tracer activity in the region of SVS. The clinical status, EEG findings, and outcome of all 53 patients were the same irrespective of the presence or absence of SVS tracer activity. We conclude that the mere presence of SVS in the absence of demonstrable cerebral arterial flow activity is not clinically significant and does not contradict the diagnosis of brain death.

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The definition of brain death is total and irreversible cessation of all brain function. Brain death is a diagnosis based on the clinical findings of coma, absence of cephalic reflexes, absence of spontaneous respiration, and flat electroencephalograms (EEG) (*1–6*). Cerebral vascular studies such as contrast angiograms, radionuclide tracer angiogram (TAG), and static scintigraphy are commonly used to confirm and document the clinical diagnosis of brain death (*5–15*).

The characteristic scintigraphic findings of brain death are the absence of cerebral arterial blood flow (CABF) seen in TAG and also absence of sagittal venous sinus (SVS) visualization in both TAG and static scans. These findings are accepted as good confirmatory evidence of brain death (*9–15*).

However, a frequent occurrence is a scintigraphic pattern showing no CABF, but with SVS visualization. There are disagreements among various reports as to the significance of this pattern of no CABF with SVS visualization (*11–27*). The controversy raised an important issue and produced a dilemma for the optimal care of comatose patients. Our current report is a retrospective study to review the significance of SVS visualization in the absence of CABF, based on our experience of cerebral scintigraphy performed at Boston City Hospital since 1983.

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METHOD

Patient Selection

All the radiotracer scintigraphy performed in our department between February 1983 and July 1986 for the purpose of confirmation of brain death, was reviewed. Only those patients whose TAG showed no evidence for CABF were included in this study.

Technique of Scintigraphy

The scans were performed either with a stationary or portable gamma camera. An intravenous bolus injection of 20–25 mCi of technetium-99m glucoheptonate was given for adult patients, and a dose of 50 μCi/kg was used for children. The blood flow studies (TAG) were recorded at 2 sec/frame. Immediate static images were then performed after the blood flow study in the conventional manner.

Interpretation of scintigrams. Interpretation of scintigrams was done by two nuclear physicians independently. The interpretation of the presence or absence of intracranial arterial flow depended on the arterial phase of the blood flow study, while the presence of sagittal sinus activity was determined on the findings in both the venous phase of the flow study as well as that of the static images. To determine the presence of arterial flow, special attention was focused at the locations of the anterior and middle cerebral artery regions. Only when there was localized concentration of radioactivity in these anatomic regions during the arterial phase was this interpreted as evidence for cerebral arterial flow, as opposed to the diffuse background activity due to circulation in the scalp. These patients were divided into two groups (A and B), according to their scintigraphic findings. Those patients with scans showing

evidence of radioactivity in the region of the SVS either in the dynamic part or static part of the scans, were put together in Group A. Those showing no evidence of SVS visualization were classified into Group B. The nuclear physicians also took notice of the "hot nose" sign which was made only when there was abnormal increase of activity in the nasal region or in the midline below the region or in the midline below the region of the circle of Willis, and when such increased activity was more intense than that in the adjacent carotid arteries.

In case of disagreement of interpretation between the two physicians, the difference was resolved by joint discussion with participation of a third physician until consensus was reached.

Review of patient's outcome. Patient's medical records were reviewed retrospectively to correlate the scintigraphic findings with the patient's hospital course. Autopsy reports, observations during surgery, and surgical specimen reports were also reviewed.

RESULTS

Between February 1983, and July 1986 there were 53 patients referred to our department for confirmation of brain death, whose scintigrams demonstrated no evidence of cerebral arterial flow (Group A and B). Both groups had clinical signs of brain death: deep coma, no cephalic reflexes, and no spontaneous respirations. Twenty-one of the 53 patients did not have technically satisfactory EEGs reflecting the difficulty of obtaining reliable EEGs among head trauma patients. The others had flat EEG tracings. The causes of brain damage included gunshot wounds, asphyxia, blunt head trauma, drowning, and intracerebral bleeding. Interobserver difference was negligible. There was only one scan which was considered equivocal for SVS visualization by one observer but was considered as positive SVS visualization by the other observer. After joint discussion, both observers agreed that there was SVS visualization.

Group A (SVS activity). There were 26 patients in this group. Nineteen Group A patients also showed positive "hot nose" signs. All 26 patients died of cardiovascular collapse and/or respiratory arrest within 1–8 days after the first scan was performed.

Group B (No SVS activity). There were 27 patients in this group. Twenty-two of these patients had positive "hot nose" signs. Twenty-six of the 27 patients died of cardiovascular collapse and/or respiratory arrest within 1–4 days after the first scan was performed. One patient survived for 17 days after the first scan. He subsequently died of cardiovascular collapse.

Examination of Brain Tissue

Group A. Four patients in this group had autopsies. In two other patients, surgical observation during craniotomy was obtained. Direct examination of the brain demonstrated diffuse softening and necrosis in all these patients. The findings were consistent with that of brain death ("respirator brain") (*29*).

Group B. Two patients had autopsies. In five other patients, brain tissue was examined during surgery. Direct examination demonstrated diffuse softening and necrosis of brain.

DISCUSSION

Brain death means total and irreversible cessation of all brain functions, thus patients with brain death will inevitably die within a relatively short period of time (days or weeks), despite intensive modern medical intervention. Establishing the diagnosis of brain death for comatose patients is neither easy nor straightforward.

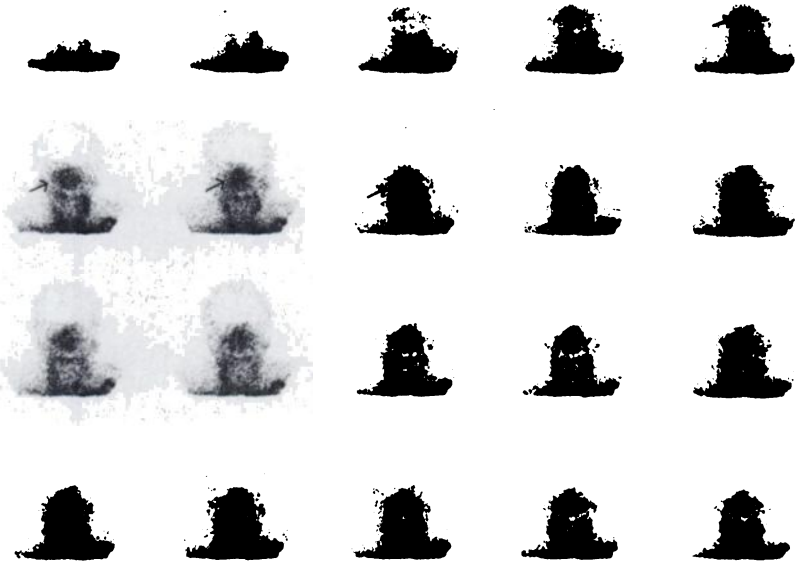

FIGURE 1
This was a 54-yr-old female patient admitted because of coma following head trauma after falling. Neurological and laboratory examinations showed signs of brain death (no cephalic reflexes, coma, apnea and two flat EEGs). Scintigrams were performed to confirm the clinical diagnosis. The dynamic scans demonstrated absence of cerebral arterial flow and positive hot nose sign (arrows).

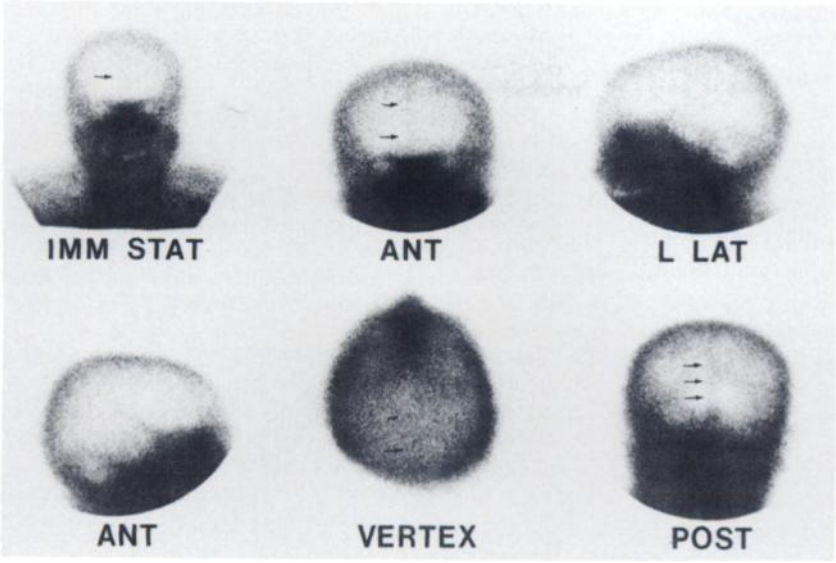

FIGURE 2
Static scans demonstrated definite tracer activity in the region of the sagittal sinus (SVSV). The patient died of cardiovascular collapse 1 day afterwards. Autopsy showed diffuse softening of the cerebrum, consistent with brain death.

Today there are more than 30 different criteria worldwide for the diagnosis of brain death. In the USA two sets of criteria are most influential and well known, the Harvard criteria, and the criteria established by the U.S. Collaborative Study Group (USCSG) (*1–3*).

The Harvard criteria rely on clinical examination and EEG results to indicate absence of brain function. In practice these indicators may be difficult to perform or unreliable to interpret. Thus, the USCSG criteria include other confirmatory tests to evaluate blood flow of the brain in determining brain death, as the brain cannot remain viable or function without blood supply (*8–11,29*). The ability to predict "imminent" death is a major component of both the Harvard and USCSG criteria. All patients fulfilling the Harvard criteria were dead within 14 days. The USCSG criteria were more liberal, extending the viable period to 3 mo (*1–5*).

The absence of blood supply may be demonstrated either by contrast angiography or radionuclide scintigraphy, which includes a tracer angiogram (TAG) and static imaging. While contrast angiography is more commonly used in Europe to confirm brain death, scintigraphy has gained wide acceptance in the USA (*26–28*). In our retrospective study, all scintigrams included showed absence of CABF. The clinical assessments of all these patients fulfilled the USCSG criteria. All patients (Group A and B) died within 8 days with the exception of one in Group B who died 17 days after the scintigrams were performed. There was no difference in outcome (death) between the two groups of patients. This study therefore confirms that SVS visualization in the absence of CABF is compatible with the diagnosis of brain death, and does not change prognosis. This conclusion was also supported by the results of (six) autopsies and surgical examination of brain tissue in seven patients showing extensive necrosis and softening ("respirator brain") (*1,2,29*).

Our explanation for why SVS visualization does not mediate against a diagnosis of brain death is twofold. First, contrast angiography has shown filling of intracranial venous sinuses through emissary veins via the external carotid circulation, despite "non-filling" of cerebral arteries (*30, 31*). This parallels our scintigraphic observations of SVS visualization and no CABF, and further supports and explains the belief that mere SVS activity is not good evidence for the presence of cerebral circulation. Secondly, the increased scintigraphic activity in the region of the sagittal sinus may not be tracer activity within the sinus itself, but rather the activity within the circulation of the vascular network of the dura and falx. This distinction is beyond the spatial resolution of the gamma camera (*32–35*). It is interesting to compare our results with that of the contrast angiogram. Among brain dead patients, the SVS was not visualized as a rule, with only isolated positive reports when SVSs were supplied by collateral emissary veins (*8,30,31*). On the other hand, the falx and tentorium are known to be very vascular structures, deriving their blood supply through the external carotid artery system (*34–36*). The frequent presence of the "hot-nose" sign (vide infra) indicating increase of external carotid collateral flow further strengthens our contention that what we considered as SVS tracer activity in our scintigrams was actually tracer activity in the falx (*37–39*).

The "hot nose" sign was first described more than ten years ago as scintigraphic evidence of internal carotid artery obstruction among fully conscious patients (23, 28). The presence of the "hot nose" sign in the TAG was due to increase in collateral blood flow from the external carotid artery through the facial and opthalmic arteries.

Among brain dead patients, cessation of internal carotid artery flow at the siphon is due to increase in

intracranial pressure, and not to intraluminal obstruction ("brain tamponade") (*8,10,30–33*). However, the hemodynamic effects are similar in patients with true intraluminal obstruction of the internal carotid arteries (*37–39*). Therefore, this same mechanism can provide the explanation for the presence of the "hot nose" sign among 73% of our brain dead patients. Although the presence of the "hot nose" sign by itself is nonspecific and may not indicate brain death, we feel that when it is present together with no CABF, it may represent a secondary scintigraphic sign to further support the diagnosis of brain death.

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