Journal of Korean Medical Science
Vol. 8, No. 4, 305-307 August 1993

# Preenhanced Computed Tomographic Findings in Brain Death

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***A patient complying with the clinical criteria for brain death was studied by preenhanced computed tomography (CT). Preenhanced CT showed apparent increased density at the base of the brain along the course of the major arterial vessels, and abnormally dense-appearing deep venous structures, like those of contrast-enhanced CT. There was a diffuse decrease in brain density with a poorly delineated ventricular system. These CT findings were very characteristic. CT as a non-invasive method seems to be valuable in the diagnosis of brain death. The relevant literature is reviewed and mechanisms showing those CT findings are discussed.***

**Key Words:** *Preenhanced CT, Brain death*

## INTRODUCTION

The basic requirements for a declaration of brain death are absence of cortical activity, absence of brainstem reflexes, and evidence that this state is irreversible (Black, 1990). Many diagnostic methods have been proposed to evaluate brain death.

They are largely classified into two types. One is demonstration of absent brain function by electrophysiologic study (e.g. electroencephalography, evoked potential studies). The other is demonstration of absent cerebral blood flow (e.g. angiography, isotope scan, ultrasound, contrast-enhanced computed tomography scan) (Black, 1990). Cerebral angiography is usually considered to be an accurate and definitive index of irreversible brain death.

Isotope angiography demonstrates absence of brain perfusion in brain death patients, is less harmful, and can be assessed by bed side imaging techniques. Transcranial Doppler may be useful in demonstrating feeble perfusion in brain death patients.

Computed tomography (CT) provides an etiological diagnosis and also demonstrates the lack of cerebral perfusion by absence of contrast enhancement of the intracranial vasculature following the injection of a bolus of contrast material (Arnold et al., 1981: Shiogai and Takeuchi, 1983; Planitzer et al., 1985).

The authors present a case of brain death in which pre-enhanced CT findings mimic normal contrast-enhanced CT. That phenomenon suggests intravascular stasis of blood flow or thrombosis reflecting absence of cerebral perfusion (Eick et al., 1981; Zilkha et al., 1982). Pre-enhanced CT may also play a role in demonstrating of cerebral perfusion in brain death evaluation.

## CASE PRESENTATION

A 15-month-old boy was transferred to Seoul National University Children's Hospital on July 5, 1992, suffering nausea, vomiting, and irritability for 2 months. He was first seen at a local hospital on May, 1992. After brain MRI and lumbar puncture, he received antibiotics for 5 weeks, under the impression of a left cerebellar abscess. However, his symptoms and signs progressed, and the size of the left cerebellar lesion increased on the follow-up brain MRI.

On admission he was irritable. Pupils were of equal size and reactive. Truncal ataxia and the left cerebellar dysfunction were noted. Cranial nerves were intact. An operation was carried out on July 13, 1992. Subtotal removal was possible and histopathological ex-

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amination confirmed the diagnosis of primitive neuroectodermal tumor (PNET). Postoperatively, he received 2 cycles of "8 in 1" chemotherapy (on August 10 and 24, 1992) and discharged with improved status. One month and ten days later (just before the third cycle of chemotherapy, on October 2, 1992), he was transferred to our emergency room with nausea, vomiting, and increasing lethargy. A large recurrent mass in the left cerebellar hemisphere was noted on follow-up CT.

His parents refused reoperation. Two weeks later (on October 16, 1992). he progressed to coma with fixed, dilated pupils. Ventilator care was started. An external ventricular drainage (EVD) catheter was inserted without response. The opening pressure was 65cm $H_2O$. Barbiturate coma therapy was not tried.

Neurological examination showed absence of brainstem reflexes (e.g. pupillary, corneal, oculovestibular, gag, and cough reflexes). Apnea test demonstrated lack of ventilatory effort even with the $PCO_2$ above 60mmHg. Electroencephalography (EEG) was taken times (12 hours, 48 hours, 7 days after he progressed to coma) which revealed barely visible waves suggesting brain death though inconclusive. About 400cc of cerebrospinal fluid (CSF) was drained for 48 hours. Thereafter the CSF was not drained any more.

CT scan, performed 6 days after his progression to coma because of cessation of CSF drainage through the EVD catheter, revealed high density along the course of the major arterial vessels which included the circle of Willis and middle cerebral arteries. Deep veins such as internal cerebral veins and straight sinus were of high density. Brain density was diminished with poor delineation of ventricular system (Fig 1). Two weeks after he progressed to coma, his heart stopped beating. Autopsy was refused.

## DISCUSSION

The absence of cerebral circulation and electrical silence in the brain are accurate indices of irreversible cerebral functional loss (Black, 1990)

The usefulness of CT in confirmation of brain death has been discussed by some authors (Arnold et al., 1981: Eick et al., 1981; Shiogai and Takeuchi, 1983; Planitzer et al., 1985). CT scan visualizes the intracranial pathology and can also diagnose the absence of cerebral blood flow by showing lack of contrast enhancement of cerebral vasculature (Arnold et al., 1981; Shiogai and Takeuchi, 1983; Planitzer et al., 1985)[1, 4, 5]. CT as a noninvasive method can play a significant role in the determination of cerebral circulatory cessation. However, reports in the literature about the role of preenhanced CT in the declaration of brain death are very rare. Only one article could be found.

Eick et al. (1981) reported that preenhanced CT examination of clinically brain-dead patients showed abnormally dense-appearing arterial structures which make up the circle of Willis, and deep venous structures, mimicking a normal contrast-enhanced study[3]. Deep cerebral venous thrombosis was proved in two cases at autopsy and angiography. But the major arterial vessels were not thrombosed at autopsy. They explained that this might be due to a diminished brain density and the exudate at the base of the brain noted at autopsy. In this case, the major arterial structure

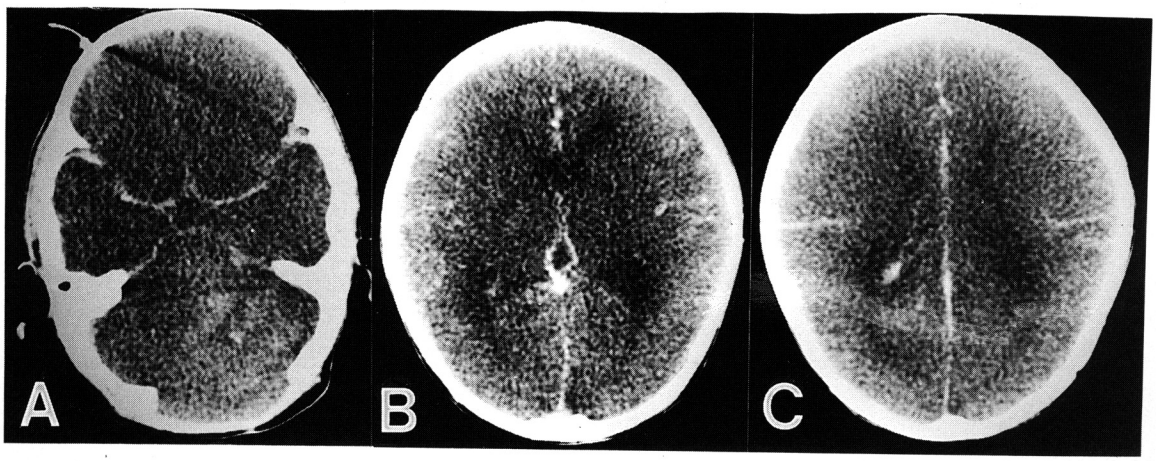


**Fig. 1.** Preenhanced CT shows apparent increased density at the base of the brain along the course of the circle of Willis and middle cerebral arteries (A), and abnormally dense-appearing deep venous structures (B, C). Diffuse decrease in the brain density and poorly delineated ventricular system are also noted.

may be also thrombosed because the shape of increased density is exactly consistent with the course of the circle of Willis which suggests an intraluminal process affecting the CT density rather than extraluminal change such as exudate in the subarachnoid space. However, the possibility of extraluminal change could not be excluded because the increased density was visible in the two neighbouring 5-mm CT sections which seemed thicker than the vessels in spite of partial volume effect. The wide distribution of increased densities, even in the interhemispheric fissure and the sulci at the high convexity like 'pseudo-subarachnoid hemorrhage', also supports the latter view.

We can not make definite documentations about the pathological status and the extent of the thrombosis because autopsy was not performed. However, high density of all the major vessels on preenhanced CT in concert with brain death and absence of CSF drainage strongly indicated the intravascular coagulation and the absence of blood flow. In conclusion, preenhanced CT as an easy and safe method can be valuable for the evaluation of cerebral circulatory arrest.

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