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Computed Tomography in Acute Posterior Fossa Infarcts

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Thirty-one cases of acute posterior fossa infarcts are reported. CT evidence of obliterated posterior fossa cisterns and hydrocephalus indicates a grave prognosis due to brainstem compression. Progressive obliteration of posterior fossa cisterns may be used as an indicator for surgical decompression. Patients with intact posterior fossa cisterns had good recoveries without surgical treatment. CT can be used to diagnose the very early phase of an acute posterior fossa infarct and has prognostic value in predicting the outcome.

Acute infarcts involving the pons, medulla oblongata, and cerebellum are associated with high mortality. Early diagnosis is essential [1–4], but has been difficult until the introduction of computed tomography (CT). We reviewed literature on this entity [1–25] and carefully analyzed 31 cases seen over the past 3 years. CT plays an important role in the diagnostic and prognostic implications of acute posterior fossa infarcts.

Materials and Methods

Over a 3 year period, we have attended at least 31 patients with acute infarcts in the posterior fossa. The 18 men and 13 women were aged 23–73 years (average, 49.9 years). Almost all patients had dizziness, lethargy, and weakness on admission. Four had sudden onset of coma without preceding symptoms. Nine had headache, and 14 had vomiting (table 1). The interval from onset of symptoms to initial CT scan was a few (4–6) hours to 2 weeks.

The 31 patients were divided into three groups according to age: (1) nine patients under 40 years old—three died, two became permanently vegetative, and the other four had good recovery; (2) 13 patients aged 40–59 years—five died and eight had good recovery; and (3) nine patients aged 60 or older—four died and five had good recovery.

Results

Five cases demonstrated isodense posterior fossas by CT (Cases 1, 2, 8, 14, and 16) a few hours to 2–3 days (case 2) after onset of symptoms. Twenty-five had hypodensity a few hours to 5 days (case 12) after the onset of symptoms. One of these 25 had mixed hyper- and hypodensities. One patient with mixed hyper- and hypodensities was initially scanned about 2 weeks after the onset of symptoms.

There were 12 fatal cases (table 1). Among these, 10 showed complete obliteration of the posterior fossa cisterns, with a small or an absent fourth ventricle. Nine of the 12 fatal cases had hydrocephalus related to swelling of the posterior fossa structures. Two had no hydrocephalus; in one of (case 5) those two, pontine and cerebellopontine angle cisterns were maintained and the perimesencephalic cisterns were obliterated.

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TABLE 1: **Summary of Cases**

Case No. (age, gender)	Clinical Findings	CT and Angiographic Findings	Results
Died without surgery:			
1 (39,F)	Lethargy and vomiting, 1 day before admission. Comatose and apneic several hours after admission. Downward nystagmus, fixed and dilated pupils. Absent doll's eye movement and corneal reflexes.	NCT: Isodensity in posterior fossa. Absence of P, CPA, and PM cisterns. Small fourth ventricle. Hydrocephalus. CCT: Nonopacification of basilar artery. Angio: Occlusion of basilar artery proximal to AICA.	Died 2 days later. Basilar artery occlusion, bilateral cerebellar infarction, and brainstem compression at autopsy.
2 (23,F)	Headache, lethargy, and vomiting 2–3 days before admission. Obtundation on admission, then comatose several hours later. Nonresponsive, flaccid, absent corneal reflex, pupils fixed and dilated.	NCT: Isodensity in posterior fossa. Small fourth ventricle, displaced to left. Absence of P, CPA, and PM cisterns. Hydrocephalus. CCT: Nonopacification of basilar artery. Angio: Partial occlusion of basilar, superior cerebellar, and posterior cerebral arteries.	Died 1 week later. Autopsy not performed.
3 (49,F)	SLE for several years. Left ophthalmoplegia, headache, and vomiting; then became comatose over 24 hr. Pupils fixed and dilated. Flaccid and areflexic.	NCT: Decreased density in brainstem and cerebellum. P, CPA, and PM cisterns and fourth ventricle obliterated. Hydrocephalus. CCT: No abnormal enhancement.	Died 4 days later. Extensive cerebellar and posterior pontine infarcts at autopsy.
4 (62,M)	Long-term hypertensive diabetic. Sudden onset of coma, pinpoint pupils, decreased right corneal reflex, no spontaneous respiration, increased right deep tendon reflex. Extensor rigidity.	NCT: Decreased density in upper pons, midbrain, and superior vermis. P and CPA cisterns and fourth ventricle normal. PM cisterns partially obliterated. No supratentorial ventricular dilatation. CCT: No abnormal enhancement.	Died 2 days later. Autopsy not performed.
5 (64,M)	Progressively increasing weakness 2 days before admission. Sudden onset of coma a few hours before admission. Nonresponsive, flaccid, and areflexic. No spontaneous respiration (intubated).	NCT: Low density in medulla, pons, and midbrain. Minimal hemorrhagic density in brachium pontis. Fourth ventricle and posterior fossa cisterns obliterated. Hydrocephalus.	Died 1 day later. Autopsy not performed.
6 (65,F)	Lethargy and weakness on day of admission (found unresponsive at home). Responded to pain by moving left side (right hemiparesis). Very weak corneal reflexes. Pupils 2 mm, weakly reactive. Eyes deviated to right.	NCT: Decreased density in brainstem and cerebellum. Posterior fossa cisterns and fourth ventricle completely obliterated. Hydrocephalus.	Died a few days later.
7 (46,F)	Hypertension for 6 years. Sudden onset of coma with preceding increasing lethargy and weakness. Pinpoint pupils. Cheyne-Stokes respiration (intubated).	NCT: Decreased density in brainstem and left and midline cerebellum and left occipital lobe. Posterior fossa cisterns and fourth ventricle not visualized. Hydrocephalus.	Died 4 days later.
8 (49,M)	Sudden onset of coma with decerebrate posturing. Pupils 2 mm, slowly reactive. Right 6th cranial nerve palsy. Responded to pain with decerebrate posturing.	NCT: Decreased density in pons and midbrain. Posterior fossa cisterns and fourth ventricle not visualized. Hydrocephalus.	Died 2 days later.
9 (53,M)	Sudden onset of coma. Nonresponsive to stimuli. Areflexic, flaccid. No spontaneous respiration.	NCT: Isodensity in posterior fossa. Hydrocephalus. Posterior fossa cisterns and fourth ventricle not visualized.	Died 2 days later.
10 (38,M)	Syncopal episode during cardiac catheterization. Progressive obtundation with subsequent coma several hours later. Responsive to deep pain with decerebrate posturing. No corneal or gag reflexes or doll's eye movement. Pupils fixed and dilated.	NCT: Decreased density in right cerebellum. Obliteration of posterior fossa cisterns and fourth ventricle. Hydrocephalus. CCT: Nonopacification of basilar artery.	Died same day.

11 (59,M)	Sudden onset of dizziness, vertigo, and headache 1 day before admission. Unsteady gait, falling to right. Right facial weakness and quadriparesis. Remained stable for 1 week, then became comatose, flaccid, and areflexic.	NCT: Decreased density in right cerebellum with displacement of small fourth ventricle to left. P, right CPA, and PM cisterns not visualized. Left CPA cistern small. Mild hydrocephalus.	Died 2 days after onset of coma.
12 (73,M)	Falling, with increasingly unsteady gait for 5 days before admission. On arrival, comatose with constricted, nonreactive pupils. Responsive to deep pain with decerebrate posturing. Left corneal reflex absent.	NCT: Decreased density in vermis, midbrain, and upper pons. Posterior fossa cisterns absent. Small fourth ventricle. No hydrocephalus.	Died 2 months later. Basilar artery occlusion and infarction of brainstem and cerebellum at autopsy.
Recovered with surgery:			
13 (26,F)	Dizziness, vomiting, and falling to right. Rotary nystagmus, right facial palsy, left hemiparesis, decreased sensation in right 5th cranial nerve distribution. Slightly decreased hearing in right ear. Obtundation 2 days later.	NCT: Decreased density in right cerebellum. Posterior fossa cisterns visualized. Ventricular size (including fourth) within normal limits. Repeat NCT: Small, compressed fourth ventricle. Small P and PM cisterns. Obliteration of CPA cisterns. Hydrocephalus. Angio: Occlusion of right posteroinferior cerebellar artery.	Survived after surgery; no deficit 6 months later.
14 (42,F)	Acute onset of nausea, vomiting, and diplopia. Right hearing loss and left sensory deficit. Subsequent coma, left hemiparesis, and bilateral 7th cranial nerve palsy 1 day later.	NCT: Small area of hypodensity in right cerebellum. Fourth and other ventricles and posterior fossa cisterns within normal limits.	Surgical decompression with removal of infarcted right cerebellum.
15 (55,M)	Sudden onset of apnea and coma. On admission, not responsive to pain; improved slightly after Narcan. Glasgow coma score 1-4-1 (minimal withdrawal). Pinpoint pupils, left conjugate gaze preference. Right hemiparesis, eyes turning left with cold caloric stimulation.	NCT: Low density in right cerebellum. Fourth ventricle compressed and displaced to left. Small P cistern. PM and CPA cisterns obliterated. Hydrocephalus.	Surgical decompression with removal of huge infarcted right cerebellum. Recovered without significant neurologic deficit.
Recovered without surgery:			
16 (31,F)	Difficulty speaking (sudden onset). Quadriparetic, became quadriplegic. Decorticate posturing later. Head deviated to left. Doll's eye movement and corneal reflexes weak.	NCT: Isodensity in posterior fossa; small fourth ventricle and posterior fossa cisterns. No hydrocephalus. Angio: Partial occlusion of basilar and left superior cerebellar arteries. Repeat NCT: Enhancement in pons and low density in left cerebellum.	Survived; slowly improved over 2 months; quadriparesis; obeyed commands.
17 (32,F)	Diabetes mellitus for years. Occipital headache, lethargy, and weakness 1 day before admission. Right homonymous hemianopsia. Disconjugate gaze. Decreased left corneal reflex. Left facial weakness.	NCT: Decreased density in left inferior cerebellum, right cerebellum, and left occipital lobe. Small fourth ventricle displaced to right. Mild hydrocephalus. Small P and CPA cisterns. NCT 1 day later, after onset of coma: Complete obliteration of posterior fossa cisterns. Infarcted areas more apparent. Progression of hydrocephalus.	Survived; nodded head and moved hands in response to questions; aphasia; quadriparesis; homonymous hemianopsia.
18 (55,M)	Occipital headache radiating to neck for 3 days. Vertigo, dizziness, and vomiting 1 day before admission. Wide-based gait. Falling to right. Nystagmus, left lateral gaze.	NCT: Decreased density in left cerebellum. Fourth ventricle displaced to right. No hydrocephalus.	Recovered; no deficit 3 months later.
19 (68,F)	Dizzy spells, unable to walk for 1 day. Ataxic, falling to left. Nystagmus on right lateral and upward gazes. Decreased sensation in left leg and trunk.	NCT: Decreased density in left cerebellum. Posterior fossa cisterns intact. Fourth ventricle compressed but not displaced.	Recovered; able to walk with walker; nystagmus on right lateral gaze 1 month later.
20 (63,F)	Right ophthalmoplegia, headache, dizziness and vomiting. Became comatose 2 days later. Corneal reflexes weak. Pupils 4 mm, reactive. Doll's eye movement present.	NCT: Decreased density in left cerebellum and brachium pontis. P, left CPA, and PM cisterns visualized. Obliteration of right CPA cistern. No hydrocephalus.	Survived with persistent quadriparesis. Alert and awake 1 month later; discharged to home.

Table 1 (cont'd.)

Case No. (age, gender)	Clinical Findings	CT and Angiographic Findings	Results
21 (54,F)	Headache, lethargy, and vomiting for 24 hr. Weakness and loss of balance. Could not walk, but could move all extremities on command.	NCT: Decreased density in vermis. Fourth ventricle compressed and displaced forward. No hydrocephalus. Posterior fossa cisterns present.	Recovered; mild weakness 2 weeks later.
22 (54,M)	Loss of balance and falling to right for 2 weeks. Left hearing loss and decreased left corneal reflex. Left 5th and 7th cranial nerve palsies. Diplopia on left lateral and upward gazes.	NCT: Patchy decreased density with minimal hemorrhagic density in left cerebellum. Fourth ventricle displaced to right. CPA cisterns small. PM and P cisterns intact. No hydrocephalus.	Recovered with hearing loss. Could walk with assistance 1 month later.
23 (25,M)	Lethargy, vomiting with aspiration pneumonia. Pupils 4 mm, reactive. Disconjugate gaze with right medial rectus palsy. Suddenly increasing lethargy; subsequent coma and atonic breathing 2 days later. Withdrawal from pain. Corneal reflexes present. Pupils smaller (2 mm) and weakly reactive.	NCT: Decreased density in midbrain with obliteration of PM cisterns. Fourth ventricle and P and CPA cisterns intact. No hydrocephalus.	Slowly recovered over one month without significant deficit.
24 (34,M)	Dizziness, lethargy, staggering gait, and vomiting on morning of admission. Unable to stand or walk. Pupils 3 mm, reactive. Corneal reflexes intact.	NCT: Decreased density in left cerebellum and brachium pontis. Fourth ventricle compressed and displaced to right. Posterior fossa cisterns intact, except right CPA cistern. No hydrocephalus.	Recovered with slurred speech. Ataxia, weakness, and dysmetria 1 month later; discharged to chronic care facility.
25 (45,M)	Lethargy, nausea, vomiting, and inability to walk. Falling to right. Pupils 4.5 mm, reactive. Corneal reflexes intact.	NCT: Decreased density in right cerebellum. Posterior fossa cisterns and fourth ventricle intact. No hydrocephalus.	Recovered without deficit 3 weeks later.
26 (53,M)	Sudden onset of dizziness. Fell; found having grand mal seizure. Disorientation, lethargy, dizziness, and weakness. Neurologic examination otherwise normal.	NCT: Decreased density in right cerebellum. Posterior fossa cisterns and fourth ventricle intact. No hydrocephalus.	Recovered without deficit 3 weeks later.
27 (64,M)	Sudden onset of vertigo, nausea, and headache, with occasional vomiting. Right-sided weakness and ataxia. Romberg test to right.	NCT: Decreased density in right cerebellum. Fourth ventricle displaced to left. Posterior fossa cisterns intact. No hydrocephalus. CCT: No abnormal enhancement.	Recovered 1 month later with mild residual weakness.
28 (65,M)	Dizziness, vertigo, and lethargy 2 days before admission. Unsteady gait with left-sided ataxia.	NCT: Decreased density in left cerebellum. Fourth ventricle compressed but not displaced. Right CPA cistern absent; P, PM, and left CPA cisterns present. No hydrocephalus.	Recovered with minimal ataxia 3 weeks later.
29 (70,F)	Lethargy, shortness of breath, and vomiting 1 day before admission. Awake and alert, but dizzy and quadriparetic. Right facial palsy and ptosis.	NCT: Decreased density in left cerebellum. Small fourth ventricle displaced to right. P and CPA cisterns intact; PM cisterns small. No hydrocephalus.	Recovered with residual dizziness and weakness 2 weeks later. Discharged to chronic care facility.
30 (31,M)	Lethargy and weakness for 2 days. Pupils 4 mm (right) and 2 mm (left), weakly reactive. Right 3d cranial nerve palsy. Falling to right. Disorientation of time and place.	NCT: Decreased density in right cerebellum. No displacement of fourth ventricle. Posterior fossa cisterns intact. No hydrocephalus.	Mild ataxia and slight confusion 1 month later.
31 (59,M)	Severe headache, nausea, vomiting, and vertigo. Left-sided paresthesia and weakness; falling to left. Left-sided ataxia. Decreased response to pinprick and vibration on right side.	NCT: Decreased density in left cerebellum. Fourth ventricle and posterior fossa cisterns normal. No hydrocephalus.	Recovered with mild left-sided ataxia. Able to walk with assistance 6 weeks later.

Note.—NCT = noncontrast CT; CCT = contrast CT; P = pontine; PM = perimesencephalic; CPA = cerebellopontine angle; AICA = anteroinferior cerebellar artery; SLE = systemic lupus erythematosus.

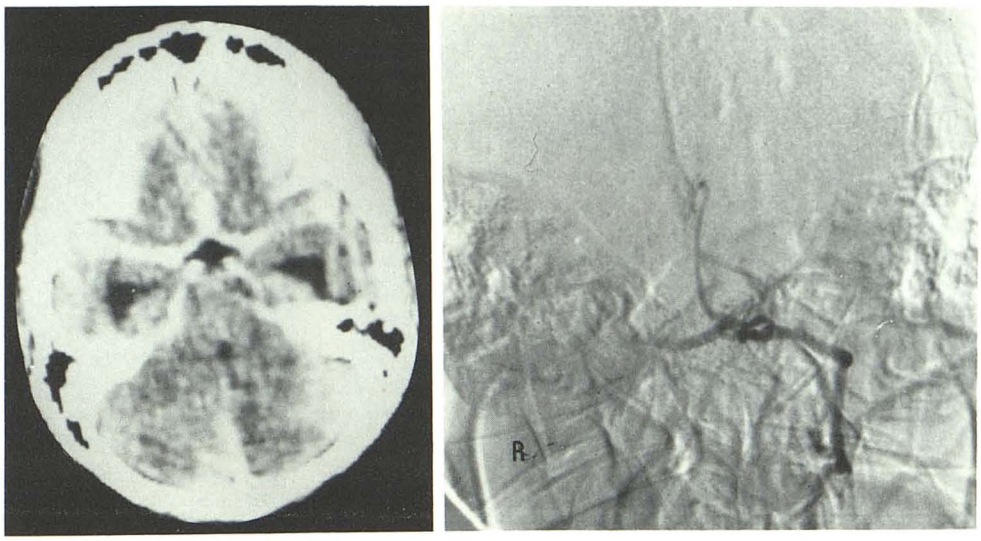


Fig. 1.—Case 1. **A**, Contrast CT. Forward displacement of small fourth ventricle and complete obliteration of pontine, cerebellopontine angle, and perimesencephalic cisterns; dilatation of both temporal horns secondary to hydrocephalus. Posterior fossa structures were isodense. Nonopacification of basilar artery. **B**, Left vertebral angiogram. Occlusion of basilar artery just distal to anteroinferior cerebellar artery. Good filling of posteroinferior cerebellar artery.

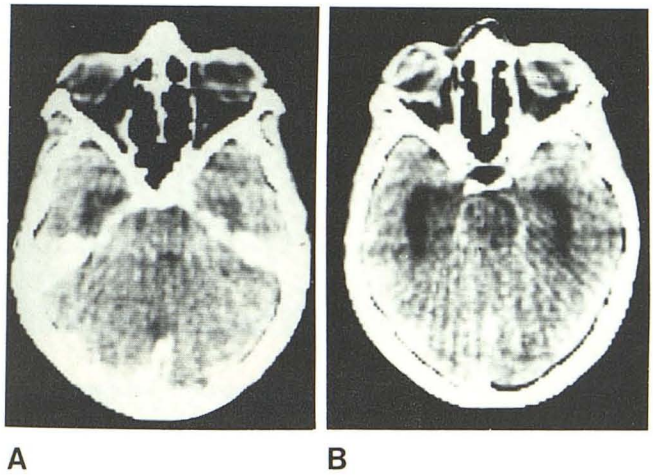


Fig. 2.—Case 2. Contrast CT. Isodense posterior fossa structures, displacement of small fourth ventricle to left and anteriorly, dilatation of both temporal horns, obliteration of posterior fossa cisterns, and nonopacification of basilar artery. Low densities within brainstem (**B**) were believed to be artifactual.

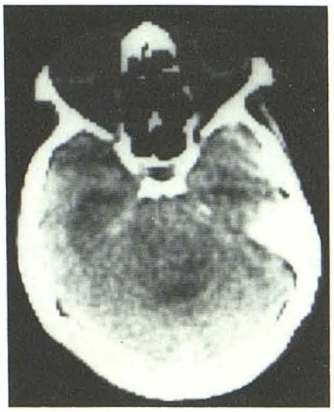


Fig. 3.—Case 3. Noncontrast CT. Low density in center of posterior fossa with complete obliteration of posterior fossa cisterns and fourth ventricle. Dilatation of both temporal horns secondary to hydrocephalus.

There were 19 survivors (table 1). One had nearly complete obliteration of the posterior fossa cisterns; another three had progressive obliteration of the posterior fossa cisterns plus hydrocephalus on the second CT examination. Three of these four patients had surgery resulting in better recovery than the one receiving nonsurgical treatment. The other 15 surviving cases had varying degrees of recovery with nonsurgical treatment. All of these 15 cases had visible posterior fossa cisterns and fourth ventricles.

The most common abnormality demonstrated by contrast CT was nonvisualization of the basilar artery.

In cases 1–3 (figs. 1–3), the cause of the posterior fossa infarct was occlusion of the basilar artery. In cases 1 and 2, CT demonstrated posterior fossa isodensity; case 3 had hypodensity in the infarcted pons and vermis. In these cases there were some common features on CT, such as complete obliteration of the posterior fossa cisterns and hydrocephalus. These three patients, who were treated nonsurgically, died.

In cases 4, 5, and 16 (figs. 4–6), the infarct was primarily in the brainstem, with less involvement of the cerebellum. In case 16, the posterior fossa structures were isodense on the first CT; an enhancing infarct of the pons was demonstrated 5 days later after angiography. The posterior fossa cisterns and fourth ventricle were not obliterated. In case 4 there were infarcts in the pons, midbrain, and cerebellum. The perimesencephalic cisterns were obliterated. The outcome in case 4 was far worse than in case 16, possibly due to more extensive involvement of the midbrain. In case 5 there was much more extensive infarction in the brainstem than in case 4. The posterior fossa cisterns and fourth ventricle were completely obliterated, and hydrocephalus

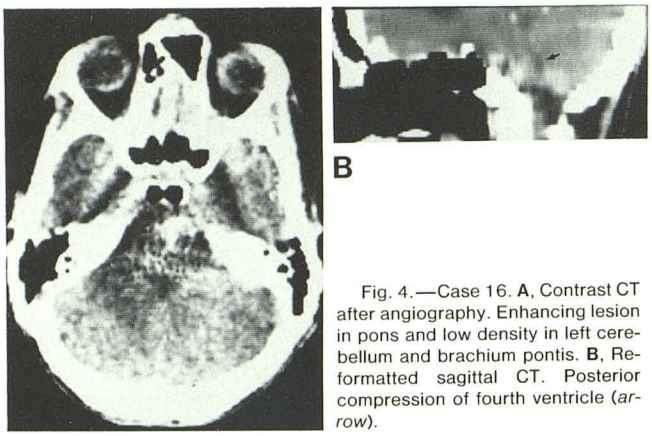


Fig. 4.—Case 16. **A**, Contrast CT after angiography. Enhancing lesion in pons and low density in left cerebellum and brachium pontis. **B**, Reformatted sagittal CT. Posterior compression of fourth ventricle (*arrow*).

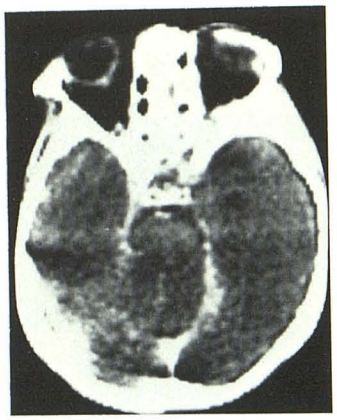


Fig. 5.—Case 4. Contrast CT. Low density in right side of brainstem and superior vermis, and partial obliteration of left ambient cistern.

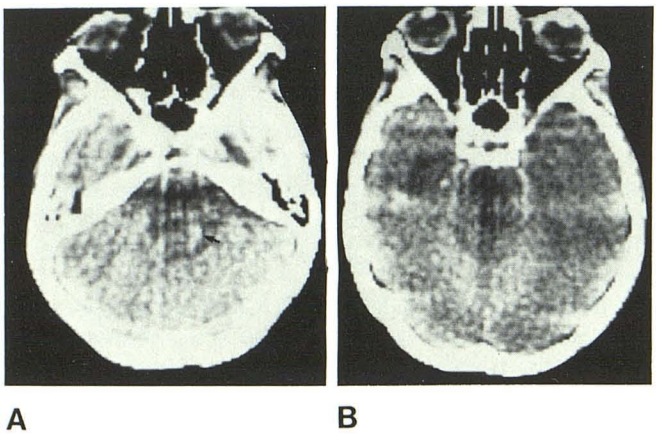


Fig. 6.—Case 5. CT before (**A**) and after (**B**) contrast. Low density in brainstem, patchy hemorrhagic density in left brachium pontis (*arrow*), and obliteration of posterior fossa cisterns.

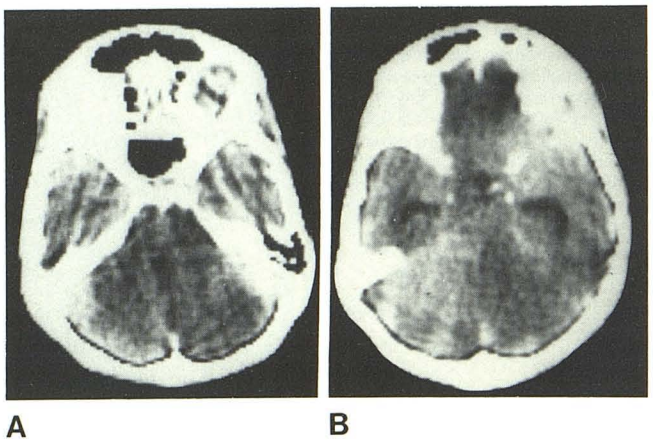


Fig. 7.—Case 17. Noncontrast CT. Displacement of small fourth ventricle to right, hypodensity of left cerebellar hemisphere, complete obliteration of perimesencephalic cisterns, small pontine and cerebellopontine cisterns, and hydrocephalus.

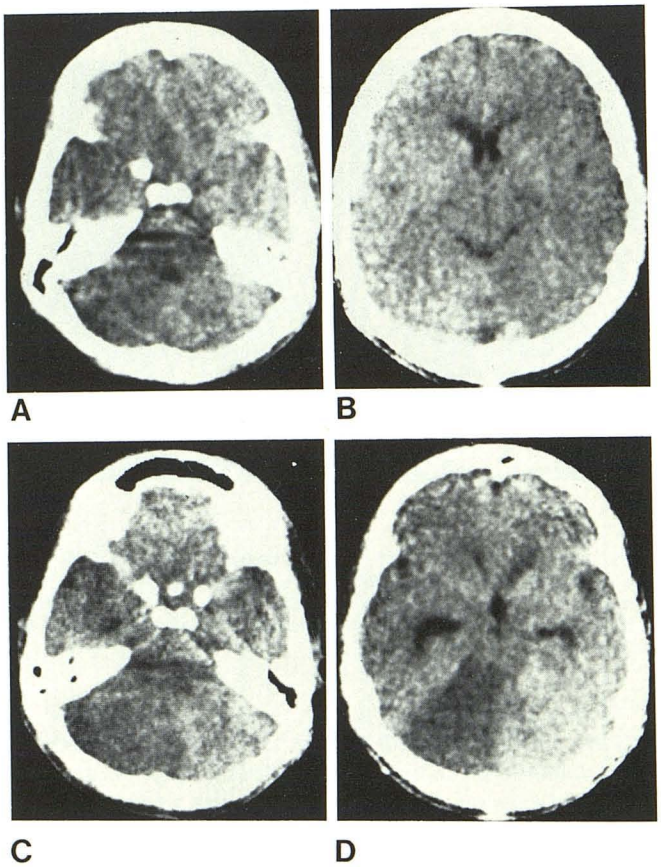
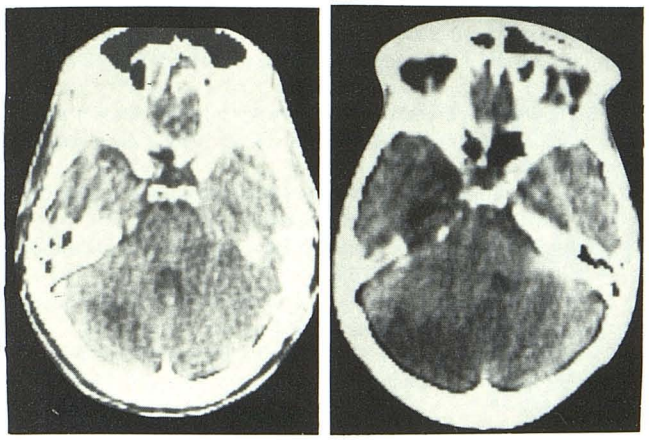


Fig. 8.—Case 13. Noncontrast CT. **A** and **B**, Slightly decreased density in right cerebellum, slight displacement of fourth ventricle to left, intact posterior fossa cisterns, and absence of hydrocephalus. **C** and **D**, Repeat scans. Complete obliteration of posterior fossa cisterns and fourth ventricle, hydrocephalus, and more distinct hypodensity in right cerebellum.

was present. These three cases demonstrated that the larger the area of involved brainstem, the worse the prognosis.

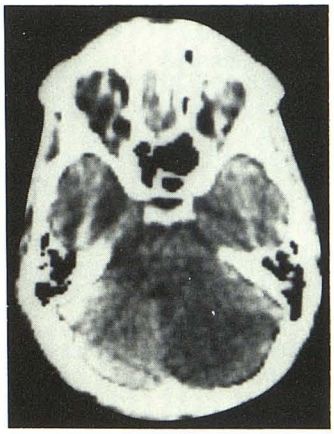
In cases 13, 14, and 17 (figs. 7–9), the initial CT showed varying degrees of infarction in the posterior fossa. In case 17 there was also an occipital lobe infarct. Initially the posterior fossa cisterns were visualized and no hydroceph-



A B

Fig. 9.—Case 14. Noncontrast CT. **A**, Small area of low density in right cerebellum, intact fourth ventricle and cisterns, and absence of temporal horn dilatation. **B**, Repeat scan. Larger area of hypodensity in right cerebellum, displacement of fourth ventricle to left. and dilatation of right temporal horn.

Fig. 10.—Case 18. Noncontrast CT. Large area of low density in left cerebellum, displacement of fourth ventricle to right, and intact cisterns.



alus was demonstrated. However, when the posterior fossa cisterns became obliterated and hydrocephalus developed, the patient's condition deteriorated. Cases 13 and 14 had emergency surgical removal of infarcted brain in order to decompress the swelling and thus prevent brainstem compression. These patients recovered without neurologic deficit within 1–6 months. Case 17 recovered with nonsurgical treatment, but had persistent severe neurologic deficits. In these three cases the cerebellar infarction was quite extensive; initial CT did not show cisternal or fourth ventricular obliteration or hydrocephalus. But follow-up CT did show obliteration of the cisterns and hydrocephalus that correlated with the progressive brainstem compression.

Surgical decompression is a life-saving procedure. Although case 17 showed survival without surgery, the result was a persistent vegetative state. In case 18 (fig. 10), the infarct in the cerebellum was also very extensive, compressing the fourth ventricle. Although there was very mild early brainstem compression, there was still enough space available for expansion of the infarcted cerebellum. Injury to the brainstem was not demonstrated. The outcome of this patient was excellent, with no surgical treatment required.

Five patients (cases 1, 2, 13, 14, and 16) had vertebral angiography. Occlusive changes were noted in each patient.

Discussion

Acute posterior fossa infarcts may result from occlusion of the vertebrobasilar artery and/or its branches [2, 3, 6–9, 14–19]. The clinical diagnosis of posterior fossa infarct may be difficult. Cerebellar infarcts are often associated with and complicated by infarcts of the lateral brainstem (which is supplied by the same arteries). The manifestation of cerebellar disturbance has often been overshadowed by the more obvious brainstem dysfunction [3, 7–9]. Angiographic diagnosis may also be difficult in the presence of isolated occlusion of a small arterial branch. Acute cerebellar infarcts and accompanying cerebellar swelling may result in brainstem compression; a large cerebellar infarct may require surgical decompression in order to reduce injury to the brainstem [4, 7, 8, 10–13, 20–25]. However, there are reports of good results after acute cerebellar infarcts without surgical intervention [2, 14, 17]. Surgery is certainly not indicated in acute brainstem infarction. Therefore, accurate diagnosis of the type and size of the posterior fossa infarct is essential for clinical management. In our experience, CT is often useful in differentiating cerebellar from brainstem infarcts.

The mortality rate seems to increase only slightly with increasing age: 34%, group 1; 38.5%, group 2; and 44.5%, group 3. However, the combined rate of mortality and poor recovery (persistent vegetative states) for group 1 was 56%, indicative of grave prognosis even for the younger patients. In reviewing the CT findings, the younger patients tended to have more associated mass effect than the older patients.

Although angiography is capable of demonstrating vascular occlusions (cases 1, 2, 13, 14, and 16) [6, 18], the precise site and the size of the infarct was often not clearly identified without CT. CT can demonstrate very early infarcts in the posterior fossa [5]. The fourth ventricle is a good landmark in locating a cerebellar infarct in those cases with isodense infarcts. In cases 1 and 2, displacement of the fourth ventricle was the major finding of cerebellar infarction (other than the nonvisualization of the posterior fossa cisterns). In case 17 there were infarcts on both sides, but the fourth ventricle was shifted away from the larger infarct.

In cases of large or massive infarction, the posterior fossa cisterns and the fourth ventricle were obliterated. Nonvisualization of the posterior fossa cisterns may be correlated with clinical manifestations of brainstem compression [11, 13, 26–28]. Obliteration of the posterior fossa cisterns plus hydrocephalus were CT signs of a grave prognosis, (cases 1–3 and 5, table 1). Progressive obliteration of the posterior fossa cisterns indicated that there was increasing brainstem compression (cases 13, 14, and 17). This may be an indication for surgical intervention to prevent death or vegetative states from brainstem injury [4, 21, 22, 26–28].

In those cases with intact posterior fossa cisterns and no hydrocephalus by CT, there was good recovery with non-

surgical treatment. These CT findings may be used in the clinical monitoring for nonsurgical management (table 1).

We believe that CT should be promptly used for the diagnosis of acute posterior fossa infarcts. These patients should be closely monitored by CT to assist in directing the clinical management in order to prevent grave complications.

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