

Posttraumatic Cerebral Infarction Diagnosed by CT: Prevalence, Origin, and Outcome

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Posttraumatic cerebral infarction is a recognized complication of craniocerebral trauma, but its frequency, cause, and influence on mortality are not well defined. To ascertain this information, all cranial CT studies demonstrating posttraumatic cerebral infarction and performed during a 40-month period at our trauma center were reviewed. Posttraumatic cerebral infarction was diagnosed by CT within 24 hr of admission (10 patients) and up to 14 days after admission (mean, 3 days) in 25 (1.9%) of 1332 patients who required cranial CT for trauma during the period. Infarcts, in well-defined arterial distributions, were diagnosed either uni- or bilaterally in the posterior cerebral (17), proximal and/or distal anterior cerebral (11), middle cerebral (11), lenticulostriate/thalamoperforating (nine), anterior choroidal (three), and/or vertebrobasilar (two) territories in 23 patients. Two other patients displayed atypical infarction patterns with sharply marginated cortical and subcortical low densities crossing typical vascular territories. CT findings suggested direct vascular compression due to mass effects from edema, contusion, and intra- or extraaxial hematoma as the cause of infarction in 24 patients; there was postmortem verification in five. In one patient, a skull-base fracture crossing the precavernous carotid canal led to occlusion of the internal carotid artery and ipsilateral cerebral infarction.

Mortality in craniocerebral trauma with complicating posttraumatic cerebral infarction, 68% in this series, did not differ significantly from that in craniocerebral trauma patients without posttraumatic cerebral infarction when matched for admission Glasgow Coma Score results. Thus, aggressive management should be considered even in the presence of posttraumatic cerebral infarction.

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Posttraumatic cerebral infarction (PTCI) is a known complication of craniocerebral trauma. A variety of mechanisms may account for this complication, including cerebral vasospasm, vascular compression, or attenuation due to adjacent mass effects producing cerebral displacement/herniation, direct vascular injury, embolization, and systemic hypoperfusion [1-13]. Infarction of the occipital pole following compression of the posterior cerebral artery (PCA) by the herniating medial temporal lobe is perhaps the most well-recognized mechanism leading to PTCI [1, 3]. The precise frequency with which cerebral infarction complicates craniocerebral trauma and its influence on mortality are not well established.

In order to ascertain this information, we retrospectively reviewed imaging studies and clinical histories of all patients with CT evidence of PTCI admitted to the University of Maryland Shock-Trauma Center during a 40-month period. The site(s) of infarction and probable etiology (based on CT, angiographic, and postmortem observations) and influence on patient mortality were evaluated.

Materials and Methods

From August 1985 through December 1988, 1332 patients were admitted to the Shock-Trauma Center of the University of Maryland Hospital with significant craniocerebral trauma.

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Indications for cranial CT included a Glasgow Coma Score (GCS) less than 15, clinical evidence of a calvarial fracture, a history of loss of consciousness following trauma, foreign body localization, and postsurgical assessment. All study patients underwent at least one cranial CT study during admission or had cranial CT performed at an outside institution before transfer to our center. A computer data base of CT records was searched for all trauma patients diagnosed with cerebral infarction during this period. All CT scans, cerebral angiograms, medical records, and autopsy reports of these patients were reviewed. A CT diagnosis of PTCI was made when distinctly marginated area(s) of low attenuation in a known arterial territory was identified on unenhanced CT scans (23 patients) and persisted on all follow-up CT scans or when sharply marginated cortical and subcortical low densities crossed arterial territories (two patients). Patients with CT evidence of diffuse cerebral infarction due to hypoperfusion (with nontraumatic causes of infarction such as a spontaneous cerebrovascular accident) or with a history of CT findings indicating infarction as possible surgical or angiographic complications were not included in the study.

The 25 patients comprised 18 males and seven females 8–91 years old (mean, 38.1) who met the criteria for PTCI. Mechanisms of injury included motor vehicle collision (11), fall (six), pedestrian–motor vehicle impact (four), blunt assault (three), and gunshot wounds (one). All initial cranial CT scans were obtained without IV contrast material using a GE 9800 scanner (Milwaukee) or a Siemens DRH scanner (Iselin, NJ) unless performed at a referring institution. A selective cerebral angiogram was obtained in one patient to document evidence of vascular injury suggested by CT findings.

All cranial CT scans and the cerebral angiographic study were reviewed by two staff neuroradiologists and the staff trauma radiologist; interpretation was recorded by consensus. Information recorded included admission CT findings, site(s) of cerebral infarction on initial or follow-up CT scans, time to recognition of cerebral infarction by CT from the date of admission, and probable mechanism of infarction based on CT and cerebral angiographic findings. Medical chart review was conducted by two neurosurgeons to ascertain admission neurologic status as reflected by the GCS, history of surgical intervention following initial CT, and patient outcome. In addition, the mortality for *all* cranial trauma patients admitted during the period of review was obtained for two groups of patients based on an admission GCS of ≤ 5 or a GCS of >5 and ≤ 10 . The results of postmortem examinations of the fixed and sectioned brain, performed in five patients, were reviewed.

Results

Sites of Infarction

Sites of cerebral infarction (Table 1) included the uni- or bilateral PCA distribution in 17 patients (Figs. 1–3), uni- or bilateral proximal and/or distal anterior cerebral artery (ACA) distributions in 11 (Figs. 1 and 2), and unilateral or bilateral middle cerebral artery (MCA) distributions in 11 patients. Among patients with ACA territory infarction, we observed five patients with unilateral and four patients with bilateral proximal ACA infarction. Eight of these patients had concurrent unilateral (six) or bilateral (two) distal ACA infarctions. In five of the 11 patients with MCA territory infarcts, concurrent infarction involving anterior cerebral and lenticulostriate distributions suggested proximal occlusion of the ipsilateral internal carotid artery. Lenticulostriate/thalamoperforating territory infarcts were diagnosed in nine patients (eight unilateral) (Fig. 1).

TABLE 1: Distribution of Posttraumatic Cerebral Infarction in 25 Patients

Vascular Territory	No.
Posterior cerebral artery	
Unilateral	12
Bilateral	5 ^a
Middle cerebral artery	
Unilateral	9 ^b
Bilateral	2 ^c
Proximal anterior cerebral artery	
Unilateral	5
Bilateral	4
Distal anterior cerebral artery	
Unilateral	6
Bilateral	2
Lenticulostriate/thalamoperforating vascular territory	
Unilateral	8
Bilateral	1
Anterior choroidal artery vascular territory (unilateral)	3
Cortical compression	2
Basilar	2

^a One with partial sparing of vascular territory.

^b Two with partial sparing of vascular territory.

^c Both with partial sparing of vascular territory.

Other areas of infarction included uni- or bilateral anterior choroidal (three) (Figs. 1 and 4) and vertebrobasilar (two) (Fig. 1) territories. In two patients, persistent cortical and subcortical uniform bands of low attenuation crossing vascular territories were compatible with infarction (Fig. 5).

Proposed Mechanism of Infarction

CT findings in 24 patients suggested that cerebral infarction was a result of focal mass effects and/or gross mechanical displacement of the brain producing transfalxine and/or transtentorial herniation (Figs. 1, 2, 4, 5). Large extraaxial hematomas with ipsilateral cerebral edema producing moderate to marked midline cerebral shift were the predominant injuries diagnosed by initial cranial CT in 17 patients. Fifteen of these 17 patients had cerebral infarctions in typical vascular distributions. Postmortem studies in three of these 15 patients confirmed the distribution of cerebral infarction(s) and demonstrated unilateral (one) or bilateral (one) medial temporal herniation. The other patient (Fig. 1) displayed left-to-right midline shift due to an epidural hematoma with left medial temporal and cingulate gyrus herniation. Infarction in this patient included the bilateral proximal and left distal ACA distribution among others.

Two of these 17 patients had cortical and subcortical bands of low density crossing arterial distributions following surgical decompression of large overlying subdural hematomas. Postmortem examination of the brain in one of these patients (Fig. 5) revealed cortical infarction in the frontoparietooccipital region adjacent to an ipsilateral overlying subdural hematoma. There was no evidence of cortical venous or dural sinus thrombosis.

Seven patients displayed multifocal cerebral contusions without significant midline shift, but with compression of basal cisterns compatible with downward transtentorial herniation.

Fig. 1.—Multifocal cerebral infarction related to epidural hematoma.

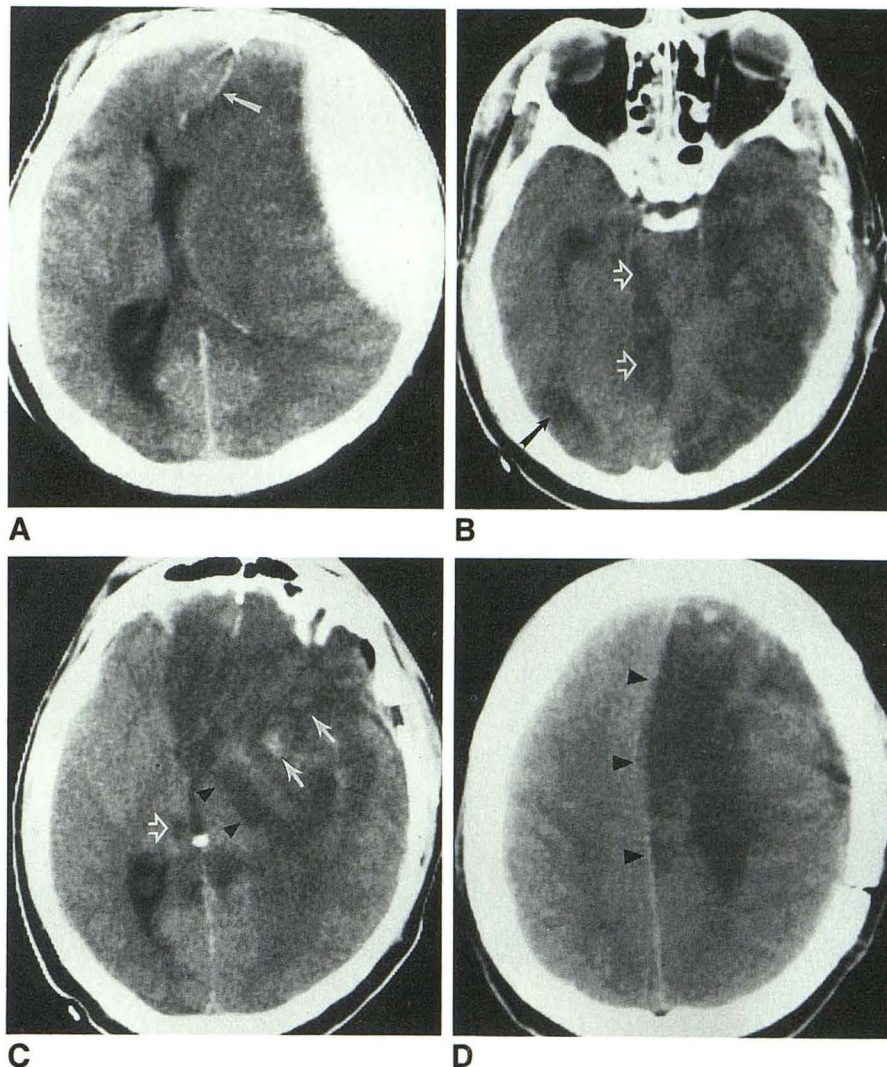
A, Axial CT image on admission in 36-year-old man after assault. Large, hyperdense epidural hematoma produces marked shift of lateral ventricles. Note bowing of anterior falx (arrow).

B, Postsurgical CT scan after evacuation of epidural hematoma reveals well-marginated low density in distribution of left posterior artery and focal low density in distribution of right posterior cerebral artery (solid arrow) suggestive of infarction. Low-attenuation area in superior vermis and right lateral rostral brainstem (open arrows) suggests infarction also. Low density in left anterior temporal lobe could result from contusion or medial temporal infarction.

C, More rostral CT section reveals well-defined low density in distribution of left anterior choroidal artery (arrowheads) and bilateral decreased density involving proximal anterior cerebral distributions, suggesting infarction. Most of distribution of left middle cerebral artery appears spared, but hemorrhagic contusion or infarction involves anterior left temporal lobe (solid arrows). Focal low density in right thalamus (open arrow) may represent infarct.

D, More rostral CT section reveals mixed anterior focal hemorrhage and low density along distribution of distal left anterior cerebral artery (arrowheads). Hemorrhagic component suggests either venous outflow obstruction or delayed reperfusion of infarcted tissue.

Postmortem examination of fixed, sectioned brain confirmed subfalcine and bilateral medial temporal herniation and verified distribution of infarcts.



Postmortem examination of the brain in one of these patients confirmed the distribution of infarction diagnosed by CT and revealed bilateral medial temporal and cerebellar tonsillar herniation.

CT in one patient with posterior MCA and PCA infarction revealed a skull-base fracture traversing the ipsilateral pre-cavernous carotid canal. Subsequent angiography revealed total occlusion of the ipsilateral internal carotid artery and a cavernous-carotid fistula (Fig. 3).

Mortality of Patients with PTCI vs General Trauma Admissions

Mortality in neurotrauma patients with complicating PTCI was compared with that in the general trauma population admitted during the same period without PTCI (Table 2). For 17 patients with PTCI admitted with a GCS of 5 or less, mortality was 76.5% compared with 64.3% for cranial trauma patients without PTCI admitted with a GCS of 5 or less ($p = .44$). Mortality was 33.3% for six patients with PTCI admitted

with a GCS >5 and ≤ 10 compared with 17.2% for cranial trauma victims without PTCI admitted with the same GCS range ($p = .28$). The likelihood of mortality among the cranial trauma population with complicating PTCI was 1.96 times that of cranial trauma patients without PTCI when matched for admission GCS; the 95% confidence limit interval was 0.78–5.13 [14]. Thus, a statistically significant increase in mortality for patients with PTCI complicating craniocerebral trauma could not be demonstrated over that of craniocerebral trauma victims without this complication when matched for admission GCS.

Discussion

A variety of mechanisms are postulated to account for the development of posttraumatic cerebral infarction [1–13]. In our series of 25 patients, CT findings either preceding or concurrent with evidence of cerebral infarction suggest that gross mechanical shift of the brain and herniation across the falx and/or tentorium accounted for infarction in 22 patients.

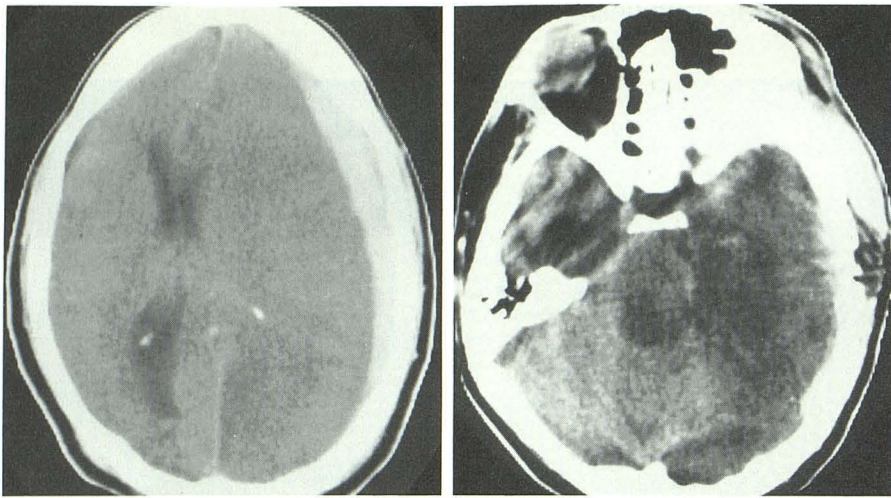


Fig. 2.—Cerebral infarction secondary to mass effect of subdural hematoma.

A, Axial CT scan in 45-year-old woman after a fall reveals large left convexity subdural hematoma producing marked left-to-right midline shift. Note complete effacement of left occipital horn. Low density is already observed in left posterior cerebral artery and focally in territory of right posterior cerebral artery.

B, CT scan 4 days after surgical evacuation reveals well-defined low density in brainstem and medial left anterior temporal lobe.

C, More rostral CT section. Low density in left anterior cerebral, left posterior cerebral, and anterior middle cerebral distributions was believed to represent infarction.

D, More rostral CT section reveals decreased attenuation involving left anterior and portion of left middle cerebral artery territory. Band of low density also involves distal right anterior cerebral distribution.

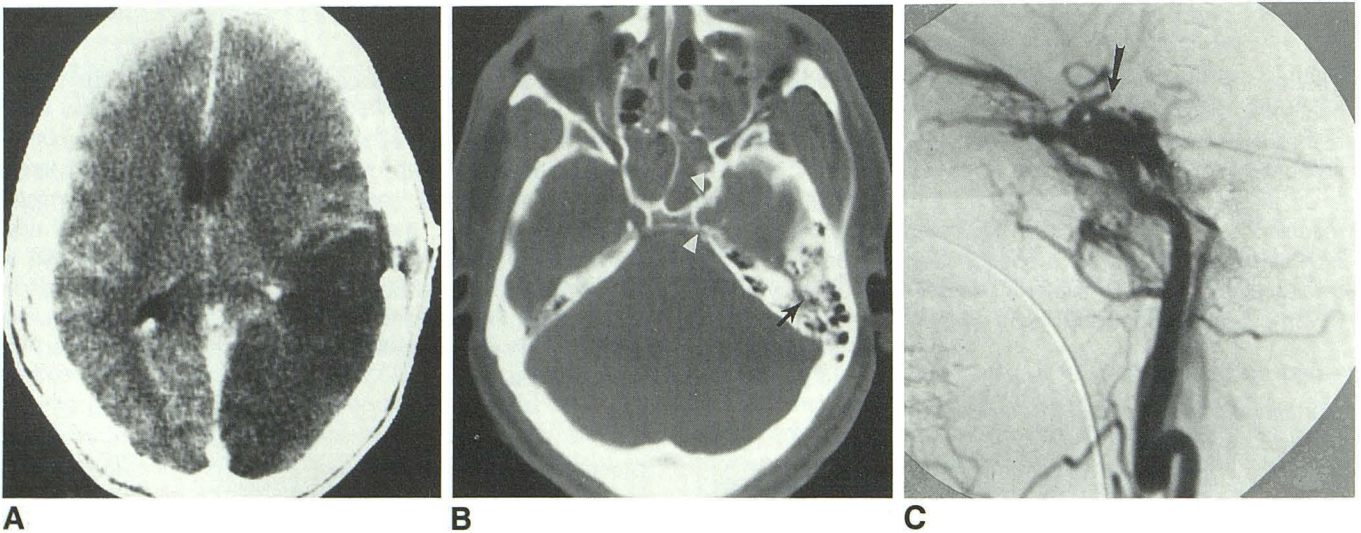
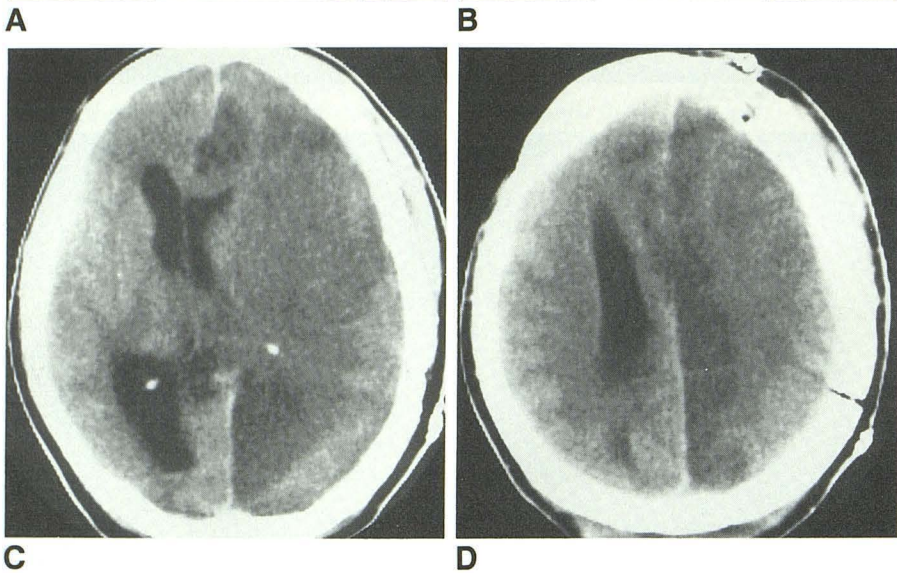


Fig. 3.—Traumatic occlusion of left internal carotid artery secondary to skull-base fracture.

A, Axial CT image in 25-year-old man after motor vehicle accident reveals wedge-shaped low density in region of left posterior cerebral and posterior left middle cerebral artery distributions. Surgical intervention in left parietal region was used for elevation of depressed fracture.

B, Axial CT scan through skull base at bone windows reveals longitudinal left mastoid fracture (arrow) and fracture crossing precavernous carotid canal (arrowheads).

C, Left internal carotid angiogram reveals complete occlusion of supraclinoid carotid artery (arrow) with carotid-cavernous fistula. Traumatic left internal carotid occlusion led to ipsilateral infarction.

Fig. 4.—Anterior choroidal infarction due to mass effect of subdural hematoma.

A, Admission axial CT scan in 91-year-old woman after a fall reveals large right convexity subdural hematoma with marked midline shift. Left occipital horn appears trapped by mass effect.

B, CT scan after surgical evacuation of hematoma. Well-defined low density involving posterior limb of internal capsule (arrows) in right anterior choroidal artery distribution was believed to represent infarction.

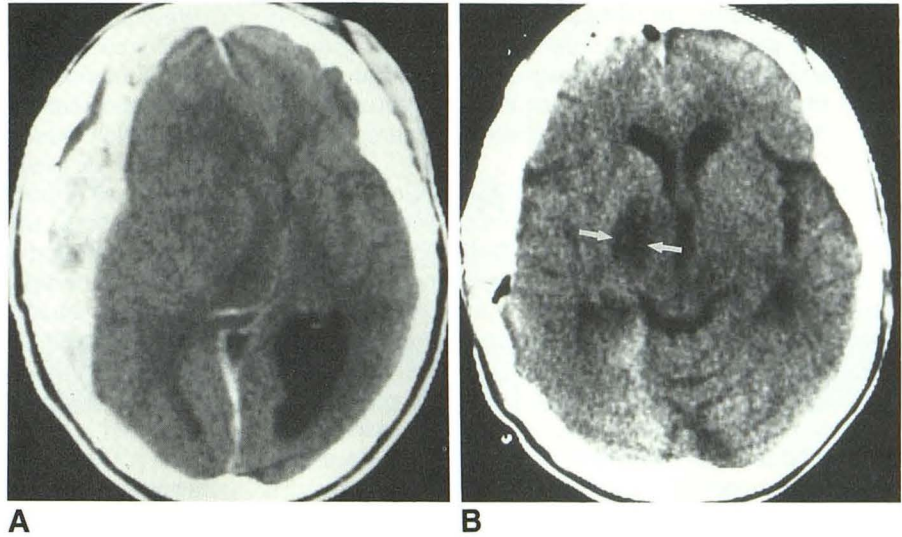


Fig. 5.—Cortical/subcortical posttraumatic infarction.

A, Axial nonenhanced CT image at midventricular level indicates diffuse right convexity subdural hematoma (arrowheads) and cortical hemorrhagic contusion with marked midline shift.

B, Axial CT image on postoperative day 6. Diffuse cortical and subcortical low density (arrows) over right frontoparietal region was believed to represent infarction related to direct pressure effects from overlying hematoma diminishing peripheral artery perfusion.

Postmortem examination of fixed and sectioned brain confirmed acute infarction of right frontotemporoparietal cortex at both gross and microscopic levels. No evidence was noted of cortical or dural sinus thrombosis.

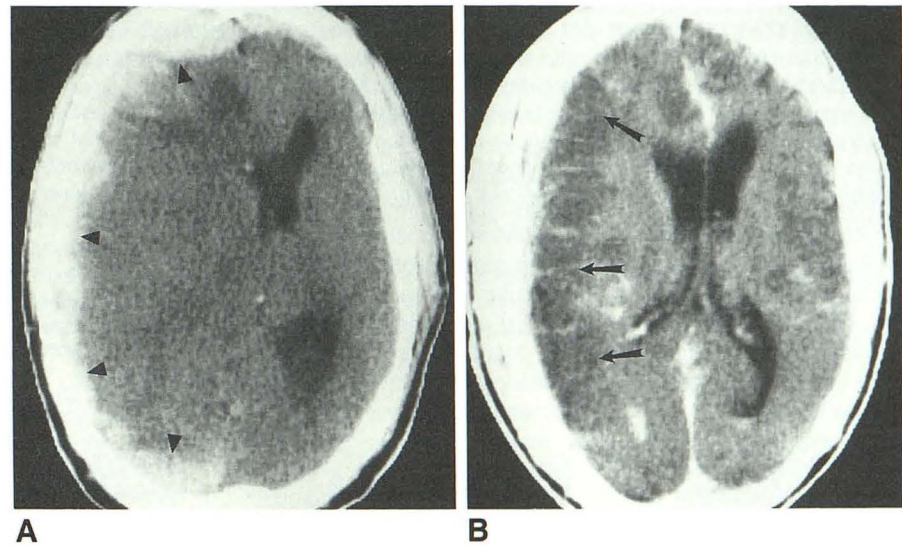


TABLE 2: Mortality in Patients with Posttraumatic Cerebral Infarction (PTCI) vs Cranial Trauma Admissions Without Complicating Infarction by Admission Glasgow Coma Scale in 25 Patients

Glasgow Coma Scale	% Mortality (No. of Patients)		Exact Test (p) ^a
	With PTCI	All Trauma Patients	
≤5	76.5 (17)	64.3 (639)	.44
>5 and ≤10	33.3 (6)	17.2 (721)	.28
>10	50.0 (2)	2.7 (7946)	Not performed

^ap = .24 for combined data for all groups using statistical guidelines of Mantel and Haenszel [14].

Infarctions due to mechanical shift were seen predominantly in the posterior and anterior cerebral vascular distributions. It is postulated [1, 3] that the PCA is compressed against the rigid edge of the tentorium by the herniating mass of the medial temporal lobe producing posterior cerebral infarction.

Sato et al. [1] described the development of posterior cerebral infarction in nine (9%) of 100 patients with CT evidence of transtentorial herniation. The infarct size and location may vary depending on the precise point of compression of the PCA and the size of the tentorial aperture [1].

A recent report [2] describes the less well recognized development of infarction in the distal ACA distribution in three patients. The authors postulate that branches of the ipsilateral callosomarginal artery may be kinked against the free edge of the falx as the medial aspect of the hemisphere herniates across the midline. The precise extent of infarction would again vary depending on how anteriorly transfalcine herniation developed [2]. Infarction in the proximal distribution of the ACA was observed in nine of our patients, including four with bilateral proximal ACA infarction. Mechanical displacement of the anterior frontal lobe may produce stretching or attenuation of proximal ACA branches leading to infarction, or conversely, downward compression of the proximal ACA or its proximal branches against the posterior ridge of the body and lesser wing of the sphenoid may lead to infarction

in both the proximal and distal anterior cerebral territories. In one of our patients with bilateral ACA infarction, cingulate gyrus herniation was found at postmortem examination. Distortion of cerebral vessels consequent to brain shift and internal herniation has been well documented neuropathologically [15].

Infarction was diagnosed in the MCA territory in 11 patients with either gross mass displacement and herniation or severe cerebral edema. Potential contributing factors include stretching and attenuation of the MCA, increased intracerebral pressure, direct pressure effects from overlying extraaxial hematomas [10], and vasospasm. Similarly, the mechanism of infarction confined to the distributions of the lenticulostriate and thalamoperforating vessels seen in nine of our patients is unclear. Presumably, these small perforating vessels could be stretched and attenuated by marked cerebral displacement leading to hypoperfusion of their vascular distributions.

Three patients in our series developed infarction confined to the posterior limb of the internal capsule, which is supplied by the anterior choroidal artery. The anterior choroidal artery may be compressed between the herniating medial temporal lobe and the cerebral peduncle medially [1]. All of the patients with infarction of the anterior choroidal distribution had massive shifts toward the side contralateral to the infarction.

The role of vascular spasm in producing cerebral infarction in our patients is unknown. Angiographically documented intracranial arterial vasospasm occurs in 5–57% of patients with craniocerebral trauma [8, 9]. Spasm is most prominent in the intradural portion of the distal internal carotid artery [6, 9]. Spasm may be due to direct vascular trauma or adjacent contusion and hemorrhage, or may be mediated by release of a vasoactive humoral factor [8]. MacPherson et al. [11] found that arterial spasm is most likely to produce ischemic damage to the cortex, but does not lead to ischemic damage of the deep gray nuclei or white matter. Since we did not perform arteriography in our patients with infarction arising from intracranial mass effects, we cannot determine what influence vasospasm had on producing infarction in these patients.

Two patients in our series developed bands of well-marginated low attenuation involving cortical and subcortical regions. These zones crossed known arterial vascular distributions. In both patients, overlying subdural hematomas produced significant mass effect on the adjacent cortex. Infarctions, particularly when hemorrhagic, may be due to direct compression of cortical veins. Postmortem examination in one of these two patients verified cortical infarction without evidence of cerebral venous thrombosis. Weisberg [10] has described infarction of brain underlying an epidural hematoma due to direct pressure effects, and underlying cerebral infarction has been reported in 8% of subdural hematomas. Presumably, direct pressure effects over the cerebral cortex result in diminished arterial perfusion leading to infarction peripherally.

Vascular injury, documented angiographically, was responsible for cerebral infarction in one of our patients with a skull-base fracture. Skull-base fractures that extend across the carotid canal can produce direct carotid vascular injury leading to occlusion, dissection with hypoperfusion and/or distal embolization, or pseudoaneurysm formation [12]. Such arterial injuries may present with either complete or fluctuating neurologic deficits, which are unaccounted for by findings on initial unenhanced cranial CT studies [5, 13] and suggest the need for immediate cerebral arteriography, particularly when skull-base fractures traverse the region of the carotid canal.

In summary, PTCl occurred in 1.9% of a large population of neurotrauma patients. Infarction was primarily due to gross mass effects producing transfalcal and/or transtentorial herniation and occurred in a wide variety of vascular territories. However, mortality for patients sustaining PTCl was not statistically significantly higher than that of craniocerebral trauma victims without PTCl admitted within a similar range of GCSs. This result suggests that the development of PTCl reflects severe cerebral injury already associated with a high mortality, but also suggests that aggressive management of the cerebral injury should not be abandoned despite the appearance of this complication.

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