

## TRAUMATIC LACERATION OF PERICALLOSAL ARTERY RESULTING IN INTERHEMISPHERIC SUBDURAL HEMATOMA: A CASE REPORT

John J. Gartman, Jr, MD, Eliot A. Atstupenas, BA, Dennis G. Vollmer, MD, Stephen K. Powers, MD

Division of Neurosurgery, University of North Carolina, Chapel Hill, North Carolina

Reprint address: John J. Gartman, MD, Division of Neurosurgery, CB#7060 UNC-School of Medicine, Chapel Hill, NC 27514-7060

□ **Abstract**—Interhemispheric subdural hematoma (IHSDH) usually manifests itself in a delayed fashion after trauma as a slow neurological deterioration with signs of the falx syndrome (paresis or seizures of the lower extremity contralateral to the hematoma). Several etiologies of IHSDH have been described, with tearing of bridging veins being especially associated with trauma. We present the second reported case of IHSDH due to traumatic arterial laceration. This lesion demonstrates a benign and delayed initial presentation. Conservative observation has been employed in the past with mixed results. Patients receiving early definitive surgical treatment have a lower mortality rate than those treated similarly having convexity SDHs. This review of 31 cases discusses the salient aspects of this clinical entity, emphasizing the need for prompt diagnosis and treatment, despite the patient's seemingly stable neurological status.

□ **Keywords**—subdural hematoma; interhemispheric; anterior cerebral artery; falx syndrome; trauma; head injury

### INTRODUCTION

Interhemispheric subdural hematoma (IHSDH) represents a rare form of subdural hematoma (SDH). Study of this usually traumatic lesion demonstrates consistencies within previously described cases relative to the mechanism of injury, time course in clinical signs of presentation, and natural history. These characteristics appear to separate it into a distinctive type of SDH. We present a case of IHSDH and describe peculiarities of this intracranial mass derived from a review of the literature.

### CASE REPORT

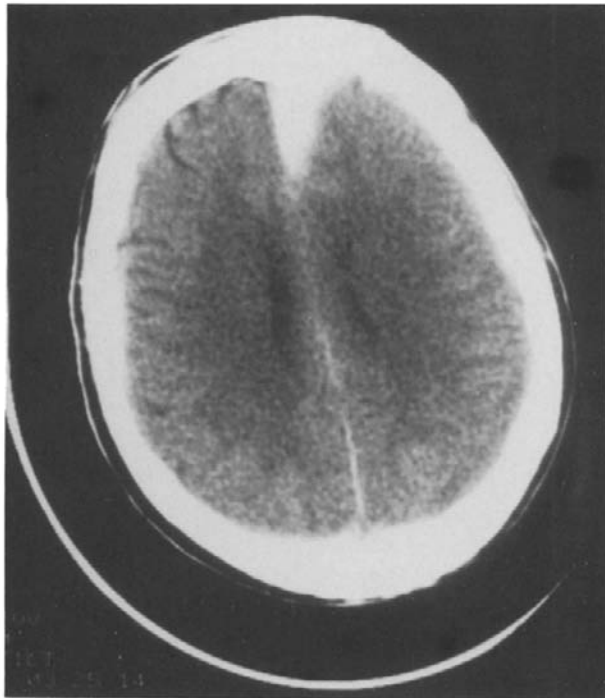
E. I., a 34-year-old female, was an unrestrained passenger involved in an automobile accident. By report, at an outside institution she was responsive, alert, and had a normal neurological exam. A history of loss of consciousness was unobtainable secondary to alcohol intoxication (plasma ethanol level=460mg/dL). A head computed tomography (CT scan) reportedly revealed a left-sided interhemispheric hematoma. (A CT scan in this situation can be supported by a recent article by Feuerman [1]. They recommend CT scanning for all patients with a minor head injury [Glasgow Coma Scale <15] and altered mental status, or hemispheric neurological deficits.) She was transferred via air ambulance, arriving at our hospital approximately six hours postinjury.

#### *Examination*

Initially, she was lethargic but awake. She opened her eyes spontaneously, followed commands with all four extremities and was conversant but confused (Glasgow Coma Scale of 14). CT scan showed an anteriorly located, left interhemispheric subdural hematoma, without midline shift (Figure 1). The hematoma was relatively small and not enlarging, so close observation without intracranial pressure monitoring was felt to be appropriate. She was under close observation and her condition remained unchanged. Then approximately eight hours postinjury, over a matter of minutes she became confused, then unresponsive and experienced an acute respiratory arrest. She was intubated, hyperventilated, and given 50 gm of mannitol.



(a)



(b)

**Figure 1.** CT scan approximately six hours post-injury (patient GCS = 14). (a) The left frontal interhemispheric subdural hematoma is seen and the midline is not deviated. (b) Coronal view shows the left interhemispheric hematoma with depression of the corpus callosum and effacement of the lateral ventricle.

A repeat CT scan showed expansion of the hematoma posteriorly, with extension over the left frontal convexity and significant mass effect (Figure 2).

### *Operation*

An emergent left frontoparietal craniotomy was performed with removal of the convexity hematoma. The interhemispheric portion of the hematoma was completely evacuated, followed by brisk bleeding. A laceration of the pericallosal artery on the medial aspect of the left frontal lobe and against the free edge of the falx cerebri was identified as the source of bleeding and was coagulated and clip ligated. It appeared that the laceration resulted from direct injury to the vessel by the edge of the falx that had torn the vessel and disrupted the integrity of the surrounding cingulate gyrus.

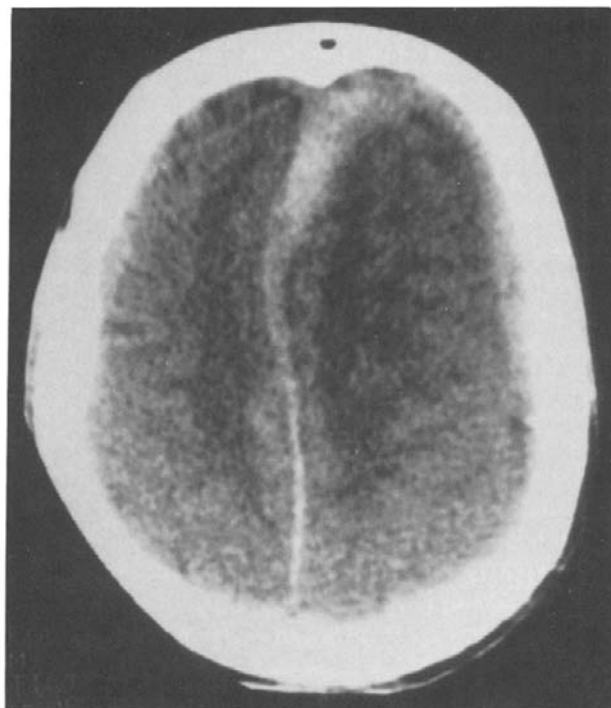
### *Postoperative Course*

She was initially noted to have 3 mm nonreactive pupils, intact gag and corneal reflexes. Two hours postoperatively her condition deteriorated, with 6 mm fixed pupils, absent corneal, doll's eyes and gag reflexes. A subsequent CT scan revealed a large pontine hemorrhage (Figure 3). Her condition never improved and she expired approximately 36 hours postinjury.

## DISCUSSION

Acute subdural hematomas (ASDHs) have long been recognized as the most common and most lethal of traumatic intracranial mass lesions. The majority of ASDHs have a definite predilection for occurring over the convexities or on the basal surface of the frontal or temporal lobes. A much rarer variety of acute ASDH occurs between the cerebral hemispheres, in the interhemispheric fissure. Several similarities in the cases of IHSDH reported in the literature warrant further discussion and appear to separate this lesion as a distinct entity within the various intracranial traumatic mass lesions.

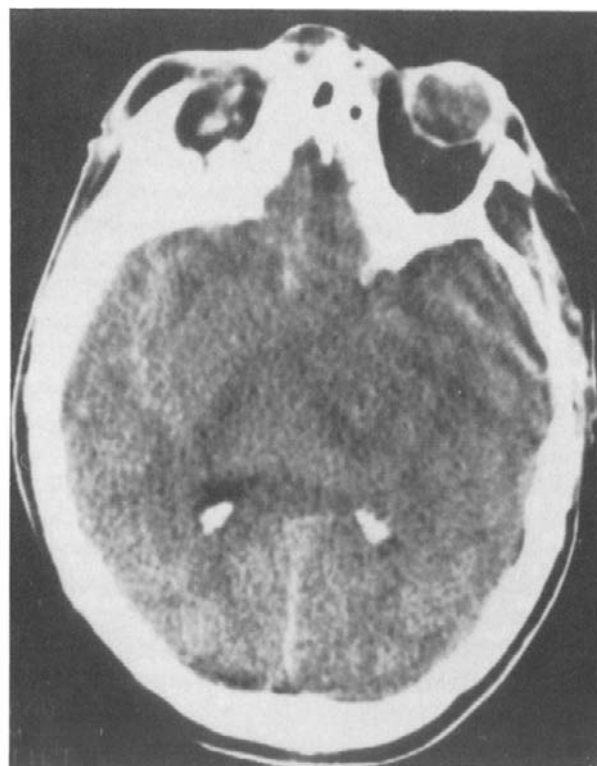
The rapidity with which ASDHs are surgically evacuated has been shown to have a significant effect upon improving the patient's ultimate outcome (2). Presumably this shortens the amount of time during which the intracranial anatomy is distorted and exposed to elevated intracranial pressure, which causes focal tissue ischemia with or without brainstem compression. Despite aggressive attempts to minimize the time elapsed between injury and surgical interven-



(a)



(b)



(c)

**Figure 2.** CT scan approximately eight hours postinjury after patient suffered acute respiratory arrest. (a and b) There is an increase in hematoma size, with extension posteriorly and marked midline shift. (c) More inferior cuts show compression of the basilar cisterns and no evidence of hemorrhage.



**Figure 3.** CT scan 2 hours postoperatively demonstrates a hemorrhage in the pontine tegmentum.

tion, traumatic ASDH has remained a devastating injury. Overall mortality in most large series of ASDH reported is over 50% (3).

Our review of 31 patients has shown a somewhat lower mortality rate of 32% for IHSDHs. A significant number of these deaths (40%) actually represent postoperative complications, not specifically related to head trauma. These results imply that IHSDHs are a more benign traumatic lesion than acute ASDHs of other locations.

The first description of IHSDH was published by Aring (4) in 1940, and the first correct diagnosis of IHSDH in life was made by Jacobson in 1955 (5). Since then our review of literature has produced 28 separate cases described in sufficient detail for a comparison and analysis (Table 1). By far, the most common cause has been trauma, and this more specifically has a distinct predilection for the occipital area. Other authors have commented on the apparent minor severity of the head trauma (6,7). Toffol et al., note that in the nine cases reviewed, none lost consciousness at the time of injury (7).

The cause of most traumatic ASDHs is the tearing of bridging veins with subsequent hemorrhage into the subdural space. It has been proposed that, be-

cause the bridging veins in the region of the interhemispheric fissure run in an anteromedial direction to the superior sagittal sinus, a blow to the occiput would provide a large shearing and stretch force upon these vessels. This would result in tearing of the veins and subsequent interhemispheric clot formation (8,9). Several other causes of IHSDH have been described including rupture of a callosomarginal and of a posterior communicating aneurysm (10,11), ventriculoperitoneal shunt in the presence of minor trauma (12), shaken baby syndrome (13) and both systemic anticoagulation and von Willebrand's disease in combination with head trauma (6,14,15,16,17,18). Our case is the second in the literature to demonstrate an arterial bleeding source. At operation the arterial bleeding point corresponded with the inferior edge of the falx. We suspect that the falcine border lacerated the artery upon impact, with subsequent hemorrhage into the interhemispheric space.

"Falx syndrome", originally described by List (19) in 1955, denotes the combination of signs that are often present with an acute interhemispheric mass, including hematoma or empyema (20) or ischemia of this region by occlusion of an anterior cerebral artery. This consists of paresis or seizures of the contralateral leg, sometimes with weakness of the arm or face but to a much lesser degree. Table 1 indicates that 20 of 31 (65%) cases presented with the falx syndrome. The anatomic correlate to this is the contralateral lower extremity motor cortex on the medial aspect of the hemisphere effected by the ischemia. If unaware of this ischemic syndrome, especially in the case of bilateral lesion, one might mistakenly localize the lesion to the cervical or thoracic spine when evaluating the trauma patient. Given this situation both possibilities must be thoroughly investigated. Interestingly, Clein and Bolton emphasized a very flattened affect in their patient with IHSDH which cleared immediately after evacuation of the mass (21). Although this finding has only been alluded to in one other case in the literature (10), it may be considered as further clinical evidence of localizing the lesion to the region of the frontal lobes and cingulate gyrus. Admittedly, this is a difficult finding to interpret in the acute trauma situation.

Whereas the majority of ASDHs produce neurologic deficits that may be obvious during the initial evaluation of the trauma patient, a recurrent theme in the presentation of IHSDH is a delay in appearance of a localizing neurologic deficit. The range of delay in presentation is from several hours up to ten days or more (2,9). Our case and others (6) demonstrate the potential for these lesions to produce rapid neurolog-

**Table 1. Summary of 31 Interhemispheric Subdural Hematoma Cases**

Year	Ref	Pt	Etiol.	Delay	Presentation	Diagnosis	Treatment	Result
1940	4	45M	trauma	2 days	*L-hemiparesis L-LE seizure coma	necropsy	trepine openings	expired
1955	5	23M	trauma	5 days	*L-leg paresis	angio	burr holes	recovered
1961	23	old	trauma	hours	*R-UE movement spasticity	angio	burr holes	expired (unrelated)
1964	18	74M	trauma anticoag	12–24 hrs	*L-leg paresis	angio	burr holes	recovered
1969	21	38M	trauma	24 days	dementia	PEG/angio	craniotomy	recovered
1970	17	42F	trauma anticoag	10 days	*L-hemiparesis LE > UE	angio	craniotomy	recovered
	10	73M	callosomarginal aneurysm	4 days	*R-LE paresis R-babinski HyperDTRs	angio	craniotomy	recovered —flat affect —sl. paresis
1977	14	80M	anticoag	10 days	progressive obutundation	CT scan IH + ICH	burr holes	expired (pulm. emboli)
1978	9	27M	trauma	24 hours	*leg weakness R > L, UE < LE	angio CT scan	craniotomy	recovered
	22	70F	trauma	36–48 hrs	*L-hemiparesis LE > UE; HA	angio	craniotomy	recovered
		44M	trauma	4 days	vertex HA confusion	CT scan angio	burr holes	recovered
1979	12	43F	trauma VP shunt	4 days	*paraparesis arm weakness	CT scan B-hematoma	craniotomy	recovered
	25	24M	trauma	36 hours	*L-hemiparesis LE > UE	CT scan	craniotomy	recovered
1982	6	54M	trauma	2 days	decerebrate CN III palsy	CT scan IH + SDH	trepine openings	expired (renal failure)
		62M	trauma anticoag	2–4 days	decerebrate no OC reflex	CT scan IH + ICH	craniotomy	expired (fatal arrhythmia)
1983	11	55M	PcoA aneurysm	immediate	HA, LOC CN III palsy	Angio CT scan	craniotomy w/clipping	recovered
1984	24	61M	trauma	6–8 hours	*L-LE paresis lethargy	CT scan angio	craniotomy	recovered
	8	59M	trauma	1–2 days	*L-hemiparesis LE > UE	CT scan	ICP monitor mannitol	recovered
1985	7	66M	trauma	24 hours	seizure wide gait	CT scan	Dilantin	recovered
1987	16	80M	trauma anticoag	3 days	*R-hemislegia R-seizures LE > UE	CT scan	craniotomy	expired (unrelated)
		81F	trauma anticoag	24–36 hrs	*comatose movement R > L, UE > LE	CT scan IH + SDH	craniotomy	expired (unrelated)
1988	15	55M	trauma anticoag	3 hours	*R-hemiplegia	CT scan angio	craniotomy	recovered
		40M	trauma anticoag	24 hours	*L-hemiparesis LE > UE	CT scan	craniotomy	recovered
		65M	anticoag	immediate	decerebrate coma	CT scan	none	expired

(continued)

Table 1. Continued

Year	Ref	Pt	Etiol.	Delay	Presentation	Diagnosis	Treatment	Result
		56M	trauma	7 hours	*R-hemiplegia coma	CT scan	craniotomy	expired
		35M	trauma	3 days	seizure, HA	CT scan IH + SDH	trefine openings	recovered
		77M	trauma vW dz	12 days	*L-hemiparesis LE > UE	CT scan	burr holes	recovered
		55M	trauma	5 days	seizure	CT scan IH + SDH	burr hole	recovered —hydrocephalus
1988	pres case	34F	trauma	8 hours	lethargy acute resp. arrest	CT scan	craniotomy	expired (pontine hemorrhage)
	##	73F	trauma	immediate	*L-hemiparesis	CT scan	craniotomy	recovered
	##	49M	trauma	2 days	*R-LE focal motor seizure	CT scan IH + SDH	Dilantin	recovered

\*Falx syndrome present  
L = left, R = right, B = bilateral  
HA = headache  
LOC = loss of consciousness  
LE/UE = lower/upper extremity  
PEG = pneumoencephalography  
VP Shunt = ventriculoperitoneal shunt  
OC reflex = oculocephalic reflex

## = personal communication of Dr. B. Walters  
PcoA = posterior communicating artery  
IH = interhemispheric subdural hematoma  
SDH = subdural hematoma  
ICH = intracerebral hematoma  
DTRs = deep tendon reflexes  
vW dz = von Willebrand's disease

ic deterioration after a previously stable course. Seventy-four percent of patients with IHSDH exhibited localizing neurological deficits 24 hours or longer after injury (see Table 1). Whether this represents acute decompensation due to brain edema or ischemia in the presence of a stable hematoma or rehemorrhage is unclear from the descriptions provided in the literature.

The noncontrasted CT scan remains the diagnostic test of choice for IHSDH. In the past both arteriography and pneumoencephalography have been used to establish the diagnosis of interhemispheric mass. Neither of these modalities plays a significant role at this time. The obvious exception to this occurs when aneurysmal hemorrhage is a serious diagnostic consideration (6). Despite recent advancements in high speed MRI, the advantage of this modality over routine CT scan for ASDHs remains to be demonstrated. Further, CT scan allows visualization of the bony anatomy, which is of practical importance in the setting of acute head trauma.

IHSDH presents a characteristic picture on non-contrasted CT scan (see Figure 1). When acute and unilateral, the hematoma appears as a high density midline mass that is convex to the ipsilateral side of the lesion but respects the falx and maintains the midline position medially (14). The ipsilateral hemisphere will often display the effects of a midline mass with ventricular shift and effacement of the gyri and

sulci. The clot may extend anteriorly to the crista galli and floor of the anterior fossa, or posteriorly to the torcula with clot layering out over the tentorium. Glista et al. have proposed that the acute hematoma is confined to the interhemispheric region by firm adherence of the arachnoid trabeculations between the brain and the parasagittal dura (9). Cases exist with concurrent ipsilateral convexity SDHs (6,16,18). Whether this represents clot migration or simultaneous tearing of a convexity-bridging vein with independent second clot formation is unclear. This possibility implies the need to search for a second bleeding source in the acute surgical situation. Ho et al. point out that the most important differential diagnosis of this CT abnormality in the traumatic setting is intracerebral hematoma, which may also appear as a concurrent lesion (6,14). Intracerebral hematomas may be distinguished by their round shape and are usually more peripheral when of traumatic origin.

The natural history of IHSDHs has been alluded to in several previous articles. Based upon a few lesions treated nonoperatively it has been suggested that as the hematoma ages it liquifies, leaving the interhemispheric fissure to migrate over the convexities (8,22). Fruin et al. suggest that many convexity chronic SDHs may actually originate as IHSDHs and come to be located over the convexities by the natural relocation of the mass (8). This would assume that the firm arachnoid trabeculations between the brain

and parasagittal dura that restricted the original hematoma no longer provide a barrier to the liquifying clot. Sibayan et al. presented a case of IHSDH that was operated upon in a delayed fashion and described a well-formed capsule that continued to retain the hematoma in its interhemispheric position (17). Toffol et al. describe a case of nonoperative treatment where the hematoma was shown to resolve on sequential CT scans but did not demonstrate the appearance of a convexity collection (7).

IHSDHs have been treated by several approaches. Early accounts employed burr-hole drainage and trephination with mixed results. Given the potential for disastrous intraoperative hemorrhagic complications involving lacerations of structures in close proximity to the superior sagittal sinus (6,23), recent authors have advocated formal craniotomy with wide exposure of the fissure and control over the sinus (9,24). In our opinion this represents the more conservative option, providing for maximal evacuation of the clot and exposure of the lacerated vessel.

A nonoperative approach has been described for patients with a definite diagnosis who are neurologically intact or who are not surgical candidates (8,22). This approach assumes that if intracranial pressure can be monitored and intracranial pressure and seizures can be controlled, the clot will resolve in situ or will liquify and migrate to a convexity position where it may be evacuated by a less invasive burr hole approach. This may be justified in the intact patient with a small clot in the setting of continuous monitoring and rapid access to full operating facilities. The danger of leaving a known mass lesion within the

intracranial compartment with the potential for rapid decompensation should not be underestimated (6). This point deserves additional emphasis in light of our documentation of an arterial source of hemorrhage in IHSDHs of traumatic origin. The pontine hemorrhage that we describe probably occurred as a result of herniation in the period between the accumulation of the clot and the patient's clinical deterioration. Had our patient undergone evacuation of the clot and repair of the offending vessel while she was still in good condition (GCS=14), she perhaps would have avoided herniation with subsequent pontine hemorrhage and made a full recovery.

### SUMMARY

IHSDs are a rare and more benign form of SDH. They present in a delayed fashion, most typically after occipital trauma. The falx syndrome is commonly seen at the time of presentation and is an important localizing feature. Disruption of the bridging veins is the most common etiology, however, bleeding from a lacerated arterial source can occur. Our review of the literature concludes that there is a need for prompt diagnosis and treatment, despite a patient's relatively stable neurological status.

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