

# TRACER ACCUMULATION IN A SUBDURAL

## HYGROMA: CASE REPORT

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***A surgically proven case of traumatic subdural hygroma gave a "positive" image during  $^{111}\text{In}$ -DTPA cisternography. This was probably secondary to a communication between the subdural and subarachnoid spaces.***

Reports of cisternographic studies in patients with subdural hematoma have described asymmetric absence of activity or obstruction of flow on the side of the subdural hematoma as the major finding (1-5). Accordingly, the negative or "cold" area is indirectly inferred to be the subdural collection. We here report a case in which a positive or "hot" image given at  $^{111}\text{In}$ -DTPA cisternography was confirmed to be a subdural hygroma at surgery.

### CASE REPORT

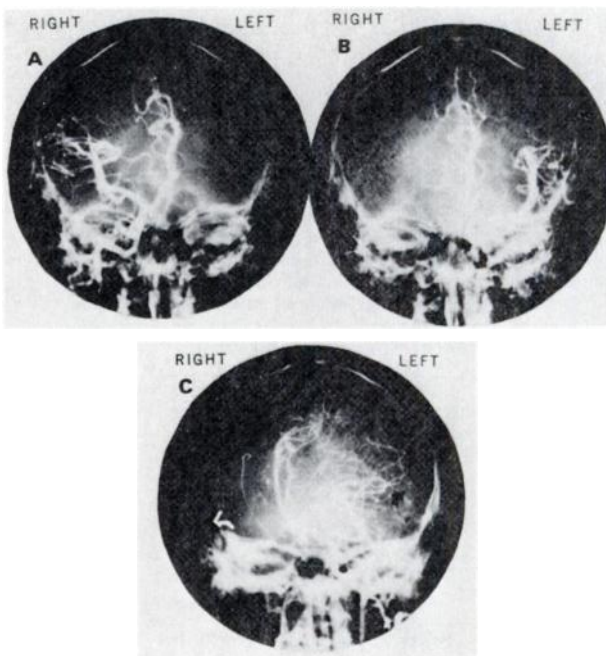
A 20-year-old man, in coma, was seen in the emergency room after being hit with a pool cue on December 18, 1974. Both pupils were fixed and dilated, the right more so than the left. Skull films showed extensive linear fractures over the right temporoparietal area. Bilateral carotid angiograms showed a 5-mm right-to-left shift of the internal cerebral veins and an 8-mm avascular mantle over the right convexity (Fig. 1A). No avascular mantle was seen over the left convexity (Fig. 1B). Craniectomy on the right side revealed contusion of the frontotemporal cortex and an acute subdural hematoma. Forty-five milliliters of subdural fluid were evacuated. There was considerable cerebral swelling. A right frontotemporoparietal craniectomy was then performed. The patient was still comatose after surgery.

Five weeks later on January 23, 1975, cisternography was performed for evaluation of possible communicating hydrocephalus. Lumbar puncture yielded clear cerebrospinal fluid with 45 mg% protein content. One millicurie of  $^{111}\text{In}$ -DTPA was instilled. Sequential scintiscans of the head and spine at 2 and 4 hr revealed that no significant tracer activity had

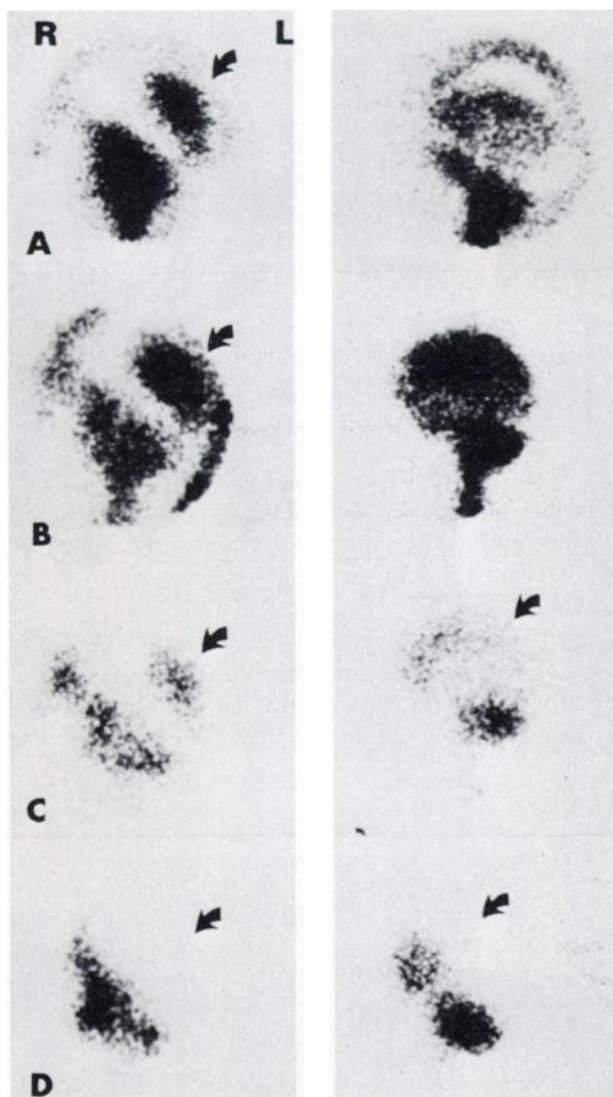
reached the basal cisterns, although the tracer could be seen in the upper spine. Nine hours after injection, activity appeared in the basal cisterns, with a large accumulation of tracer over the left convexity. Repeated projections located it at the periphery of the head. Little activity had reached the right sylvian fissure. No identifiable ventricular penetration was noted (Fig. 2A). At 24 hr (Fig. 2B), no significant

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**FIG. 1.** (A) Right carotid angiogram (12/19/74) shows 8-mm avascular mantle and 5-mm right-to-left shift of internal cerebral vein. Elevation of knee of right middle cerebral vessels is consistent with either hematoma or temporal-lobe contusion. (B) Left carotid angiogram (12/19/74) shows no evidence of avascular mantle over convexity. (C) Left carotid angiogram (1/24/75) shows 24-mm avascular mantle over left convexity (arrows) and 15-mm left-to-right shift of internal cerebral vein.



**FIG. 2.** Sequential scintiscans of head in anterior (left) and left lateral (right) positions. (A) Scan taken 9 hr after tracer instillation in lumbar subarachnoid space shows strong tracer uptake over left convexity (arrow). Uptake at base of brain (anterior view) has unusual configuration (it bends to right). It probably represents basilar cisterns and partial filling of sylvian cistern, these being distorted by mass lesion on left convexity and by surgical deformity over right convexity. There is no ventricular penetration. (B) Tracer uptake over left convexity is unchanged at 24 hr. Note gradual progression of radioactivity toward right sylvian cistern. (C) Scan taken 48 hr after instillation and 1 hr before surgery still shows no change in tracer uptake at left convexity. Physical decay of nuclide gives fewer counts. Sagittal sinus shows no activity. (D) One hour after surgery, tracer has disappeared from left convexity.

change in tracer uptake over the left convexity had occurred, nor was there further progression of activity. A bilateral carotid angiogram showed a 15-mm left-to-right shift of the internal cerebral vein. A 24-mm avascular mantle was noted over the left convexity, corresponding to the tracer collection seen on cisternography (Fig. 1C). Before surgery the next morning, at 48 hr after  $^{111}\text{In}$  injection, scintiscans of the head again showed no change in the position of the tracer over the left convexity (Fig. 2C).

Trephines were performed over the left frontal and parietal convexity. The tense dura was entered with a dural screw. Over 60 ml of straw-colored fluid, with protein content of 540 mg%, gushed out. The cortex of the brain was seen lying against the dura and appeared to be intact. No neomembrane was seen. One hour after surgery, a repeat scintiscan of the head showed complete disappearance of the tracer collection over the left convexity (Fig. 2D).

#### DISCUSSION

Here we have a documented subdural fluid accumulation in which the cisternographic examination showed a positive or "hot" image.

A subdural hygroma is an abnormal localized collection of fluid with high protein content; it may be clear, xanthochromic, or blood-tinged in appearance. It is most commonly caused by head trauma. Symptoms and signs are essentially those of extradural or subdural hemorrhage and are secondary to compression of the underlying brain. The hygroma manifests itself as an avascular mantle in cerebral angiography and cannot be distinguished from a subdural hematoma except at surgery.

The subdural fluid is generally thought to accumulate because of disruption of the arachnoid membrane, with leakage of cerebrospinal fluid into the subdural compartment. Loculation of the cerebrospinal fluid is probably due to a ball-valve mechanism involving cerebral edema. The high protein content of the cerebrospinal fluid may also be a factor in attracting the fluid into the subdural space through the arachnoid tear.

Decosta and Adson (6) reported a case of a subdural hygroma in which an arachnoid tear was observed at surgery, 3 days after a head injury. They credited Naffziger (7) as the first to observe this type of subdural fluid collection at surgery.

While Decosta and Adson and others (8-10) considered subdural hygroma to represent a clinical entity separate from subdural hematoma, Munro (11,12) considered it to represent one end of a wide spectrum of conditions encountered in traumatic subdural fluid collections, depending upon its relative content of blood and cerebrospinal fluid and the time of discovery of the lesion.

In our case, the production of a "positive" image by tracer accumulation within the subdural fluid collection suggests that a communication existed between the subarachnoid and subdural spaces and supports the current concept of the pathogenesis of traumatic subdural hygroma.

In previous reports of "positive" images of subdural hematomas in cisternography, the findings have been ascribed to other processes, such as diffusion

of tracer across an intact arachnoid membrane (13), tracer accumulation due to underlying cortical damage (1), or encephalomalacia (14). Tracer accumulation in the subdural fluid in these cases may possibly have occurred through an arachnoid tear.

## CONCLUSION

A subdural hygroma, clinically indistinguishable from subdural hematoma, presents as a "positive" image in  $^{111}\text{In}$ -DTPA cisternography. This finding lends further support to the current concept that subdural hygroma is formed by a tear of the arachnoid membrane.

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