CASE REPORT

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Post-Anesthesia Uncal Herniation Secondary to a Previously Unsuspected Temporal Glioma

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ABSTRACT: We report the case of a 21-year-old male who sustained an uncal herniation and subsequent brain death following general anesthesia, for a minor orthopedic procedure, owing to the presence of a large, unsuspected temporal glioma. The possible factors responsible for the precipitation of this event are appraised.

KEYWORDS: pathology and biology, inhalational anesthetic, uncal herniation, glioma, death

The slow growth rate of low grade astrocytomas enables these lesions to attain a significant size prior to the appearance of neurological deficits clinically. Although the intracranial cavity can accommodate a large, slow-growing mass lesion (particularly in "silent" brain areas), a point is reached where the threshold to accommodate is lost and any increase in the mass results in an exponential increase in intracranial pressure (ICP).

We present a case of a neurologically intact patient who underwent general anesthesia for a minor orthopedic procedure. Postoperatively, the patient developed a depressed level of consciousness followed by coma and subsequent brain death due to an uncal herniation secondary to the presence of a large, previously unsuspected, non-dominant temporal glioma. The possible mechanisms responsible for precipitating this acute herniation are assessed.

Case Report

The patient was a 21-year-old male who was in excellent health except for a two-year history of frontal headache. The patient had no other neurological complaints, including a negative history of seizure, and was neurologically intact on numerous exams. The patient had performed well in activities requiring a high level of motor strength and coordination up to the date of his demise. Computed tomographic images of the sinuses six months prior to the current presentation were normal; the region of the temporal lobe, however, was not visualized in this study. A diagnosis of migraine headache was suggested and no further work-up was recommended.

The patient underwent a minor orthopedic procedure on the right shoulder under general anesthesia. Induction agents included fentanyl, midazolam, propofol, and succinylcholine, while nitrous oxide, isoflurane, and droperidol were used to maintain anesthesia. Anesthetic reversal was achieved with neostigmine and glycopyrolate. The patient received 1.3 liters of crystalloid intraoperatively. Postoperatively, he was awake, alert, and oriented initially; however, several hours later he complained of a severe, throbbing headache, lightheadedness, and several episodes of nausea and vomiting. Later that evening, he was found unresponsive and cyanotic. Emergency intubation was performed. A CT scan revealed a large temporo-parietal non-enhancing, low-density lesion and uncal herniation (Fig. 1). Vigorous medical intervention, including mannitol, furosemide, dexamethasone, and hyperventilation were begun and emergency transfer to our institution for definitive neurosurgical care was arranged. The patient was brain dead on arrival. For this reason, further studies (such as magnetic resonance imaging to better define the hypodense region seen on CT as tumor/edema versus infarction) were not obtained. Autopsy was performed.

Neuropathologic Findings

Gross examination revealed generalized right hemispheric swelling, most pronounced over the right temporo-parietal area. There was evidence of massive right transtentorial and mild right subfalcine herniation. On coronal sectioning, a large, diffusely infiltrating, focally micro-cystic, gelatinous neoplasm involving the right temporo-parietal region was identified. It extended from the coronal level of the ventromedian thalami caudally to the coronal level of the trigone and posterior horns. The neoplasm involved the right temporo-parietal white matter, amygdala, hippocampus, insular cortex, claustrum, and portions of the external capsule (Fig. 2). Microscopically, a well-differentiated fibrillary astrocytoma was demonstrated (Fig. 3). The tumor was extensively sampled from different areas and coronal levels. There was no evidence of endothelial proliferation, necrosis, overt cellular anaplasia, or mitosis.

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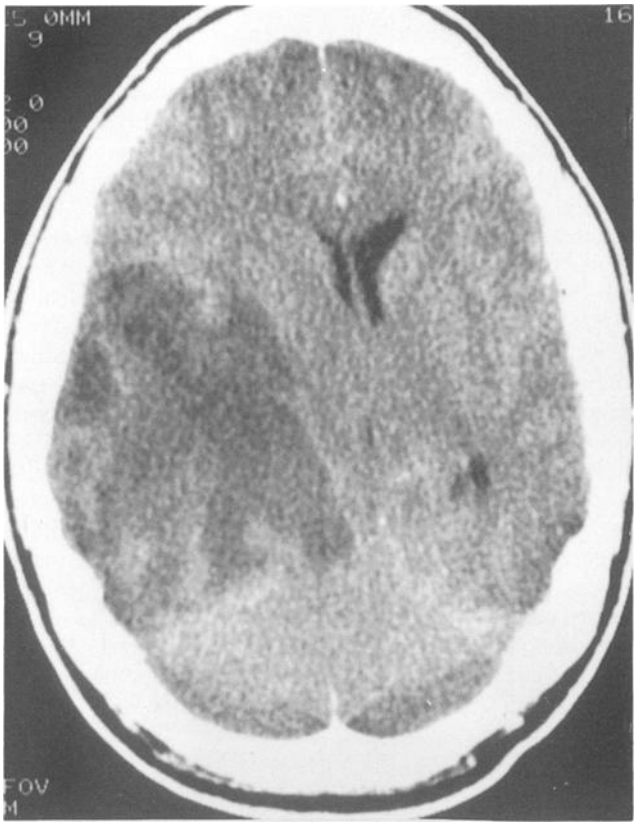

FIG. 1—*Post contrast computed tomographic scan demonstrating a nonenhancing, hypodense lesion in the right temporal-parietal region with uncal herniation and shift of the midline structures.*

Discussion

Well differentiated fibrillary astrocytomas may occur at any age but are most common in the third and fourth decades of life [*1*]. Low grade astrocytomas may attain a large size due to their slow growth rate and the ability of the intracranial contents to accommodate to a slowly evolving mass lesion prior to the appearance of clinically detectable neurologic deficits.

Intracranial pressure is a critical physiologic parameter that must be maintained within a narrow range [*2*]. The rigid confines of the skull restrict the volume of the intracranial contents. If this were strictly true, any additional volume increase would increase the ICP. In reality, however, the brain has the ability to compensate initially for increases in volume by virtue of cerebral vein compression, CSF diversion into the spinal subarachnoid space, and limited intracranial shifting. A volume-pressure curve [*3*] indicates that the intracranial pressure can be maintained at normal levels in spite of an expanding mass until the available displaceable volumes are exhausted. Beyond this point, any further increase in intracranial volume will result in an exponential increase in the intracranial pressure. When a mass lesion undergoes a rapid increase in size, the pressure-volume curve is shifted to the left and rapid increases in ICP are seen with smaller changes in volume. Therefore, the initial ability to compensate for volume-increases with absent or minimal symptoms may mask an impending, rapid rise in pressure and sudden neurologic deterioration [*2–4*]. The resulting focally elevated pressure causes a shift of the brain across a free edge of the relatively inelastic dura or into a bony foramen resulting in a herniation syndrome.

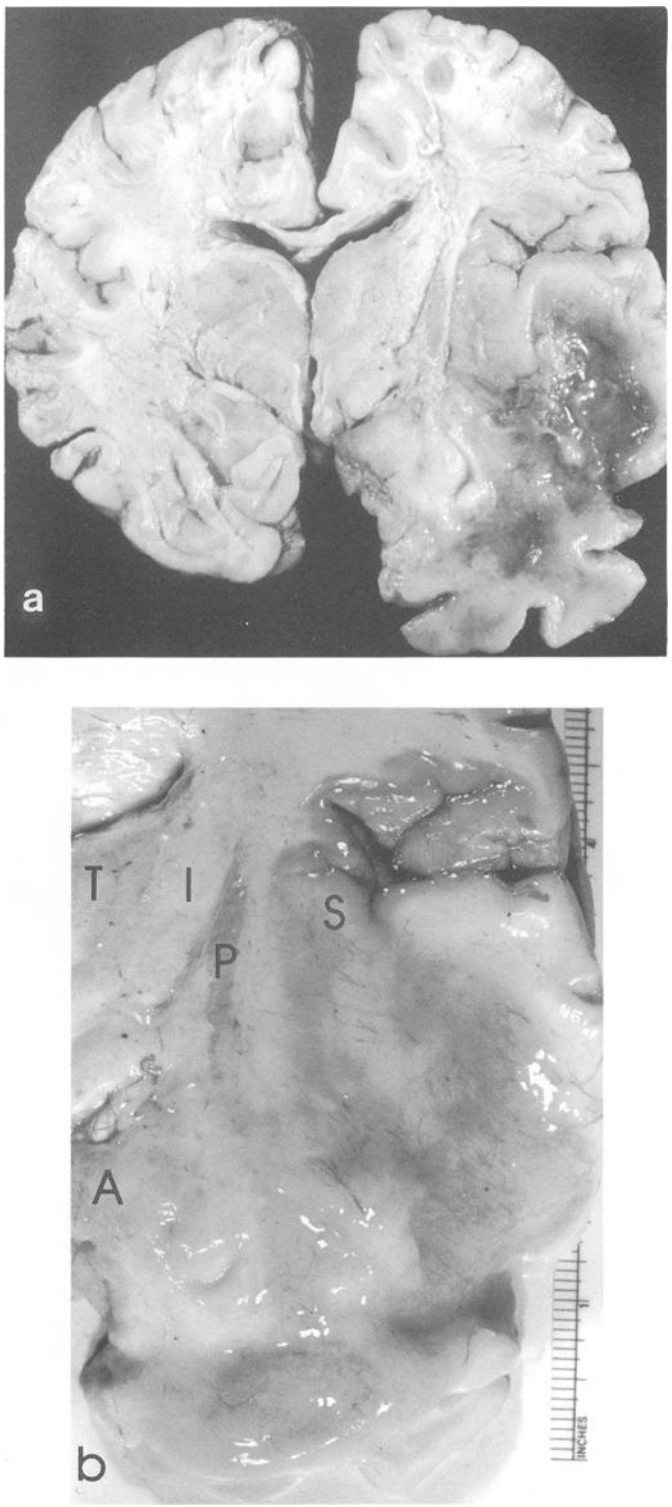

FIG. 2—(a) *Coronal section of the fixed specimen at the level of the mammillary bodies showing a massive diffuse glioma involving the convexity as well as mesial portions of the right temporal lobe;* (b) *A close-up view of a coronal section from the fresh specimen depicting a diffuse, gelatinous neoplastic process involving the right temporal and parietal lobes (A: amygdaloid complex, I: internal capsule, S: insula, P: putamen, T: thalamus).*

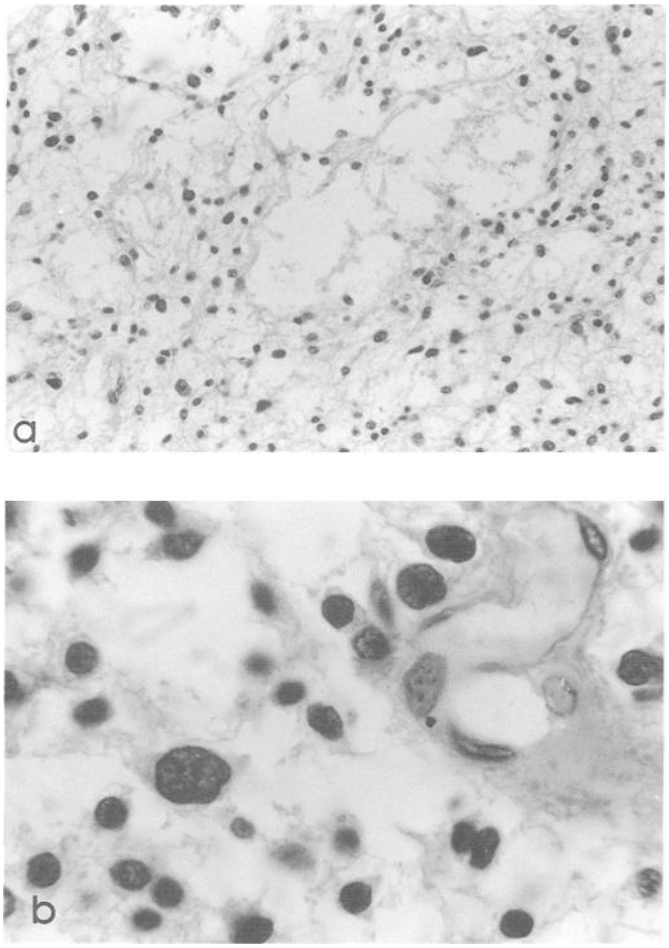

FIG. 3—(a) *Low-power view showing a well differentiated fibrillary astrocytoma with evidence of microcystic change in the center of the photomicrograph;* (b) *Atypical neoplastic glia in the vicinity of a tumor vessel. There is no evidence of endothelial proliferation. Hematoxylin & eosin (3*a *×100; 3*b *×1000).*

Brain volume may be variably increased by the presence of a tumor while it may be further significantly increased by cerebral edema and/or increases in cerebral blood volume (CBV) [*2*]. In this case, the anesthetic may have altered cerebral blood flow (CBF), with a consequent elevation in CBV, in a brain already compressed by tumor mass and intra- and peritumoral edema. In addition, hypoventilation during and/or following anesthesia probably increased the pCO_2 resulting in vasodilation and increased intracranial pressure due to an increase in CBV [*2,5*]. In the postanesthetic period, residual anesthetics can result in nausea and vomiting with subsequent spikes in ICP.

Anesthetic agents can also produce an elevation of ICP via several mechanisms. These agents, particularly the volatile anesthetics (including nitrous oxide, halothane, enflurane, and isoflurane), are vasodilators and thus increase ICP by increasing the CBV [*6,7*]. Some inhalational anesthetics can also directly affect the metabolism of CSF. Enflurane has been shown to increase the rate of CSF production while both enflurane and halothane can decrease the rate of CSF resorption [*8*]. In addition, volatile anesthetics produce a greater rise in ICP in patients with intracranial space occupying lesions than in normal controls, an effect that is unrelated to hypoxia or hypercapnia. The proposed mechanism is anesthetic induced vasodilation resulting in increased CBV [*9*]. The normal compensatory mechanisms, that is, the redistribution of CSF and venous blood, are at least partially exhausted when a space occupying lesion is present.

Transcranial doppler studies suggest a derangement of cross sectional vessel area and cerebral blood flow in response to propofol and hyperventilation in the presence of a brain tumor. Schregel et al. [*10*] have shown that the usual response to propofol administration (that is, decreased mean arterial pressure and no significant change in vessel luminal diameter) seen in a non-tumor-bearing hemisphere may be reduced, or reversed, in the contralateral, tumor-bearing hemisphere.

In the case presented, headache may have been the initial, albeit non-specific, presentation of intracranial pathology. This unsuspected space-occupying lesion diminished the patient's intrinsic ICP compensatory mechanisms placing him at a point on the pressure-volume curve at which the normally insignificant increased cerebral blood volume produced by the volatile anesthetics resulted in a sufficient rise in ICP to produce cerebral herniation. The headache, nausea, and vomiting heralded the presence of increasing ICP during the postoperative period.

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