Neurosurgical forum

Postoperative Tension Pneumocephalus

TO THE EDITOR: We wish to congratulate Dr. Ishiwata and his colleagues on their interesting article concerning tension pneumocephalus after surgical drainage of chronic subdural hematomas (Ishiwata Y, Fujitsu K, Sekino T, et al: Subdural tension pneumocephalus following surgery for chronic subdural hematoma. J Neurosurg 68:58-61, January, 1988). However, we would like to make some remarks on this subject.

Subdural tension pneumocephalus is induced by postoperative entrapment of air within the subdural space and was probably first described in 1962 by Ectors.1 The mechanism that induces this gaseous mass effect is unknown. Raggio2 does not believe that dilatation of trapped air as it warms from ambient to body temperature1 is enough to explain this phenomenon. Another cause could be a rapid increase in cerebrospinal fluid secretion following brain decompression. Computed tomography (CT) measures only x-ray absorption and not pressure. As Ishiwata, et al., wrote, there are only two methods that indicate postoperative tension pneumocephalus: either careful intraventricular pressure monitoring after surgery or the presence of resonant air spouting and bubbles blowing through the most elevated burr hole at reoperation.

The appearance of the anterior margin of the cerebral detachment on the CT scans is mainly related to the location of the slice as well as to the number of corticodural veins and the site of entry into the dura mater. These fixed points are likely to hinder the expansion of liquid effusion at first and of air accumulation later. We think that the "peaking sign" and the "Mt. Fuji sign" are not specific of tension pneumocephalus. The presence of subarachnoid air on CT scans demonstrates only arachnoidal tearing during the pre-, peri-, or postoperative period.

The real frequency of this form of pneumocephalus is difficult to establish. To prevent this serious complication, since 1962 it has been our practice to close the posterior parietal burr hole, but leave the anterior frontal burr hole open in a sterile dressing without a drain during the first 48 hours until the patient recovers neurologically. Since adopting this technique we have observed neither secondary neurological deterioration nor infection complications.1

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References

TO THE EDITOR: The recently published article by Ishiwata, et al., concerning postoperative pneumocephalus (Ishiwata Y, Fujitsu K, Sekino T, et al: Subdural tension pneumocephalus following surgery for chronic subdural hematoma. J Neurosurg 68:58-61, January, 1988) was not only of interest to me but is also timely, considering the recent trend toward more neurosurgical procedures for elderly individuals. I wish to make some comments on their paper.

Based on computerized tomography (CT) findings, the authors stressed the importance of differentiating symptomatic subdural tension pneumocephalus from the asymptomatic variety. In my experience, however, pneumocephalus without tension is not always asymptomatic. As I have found in a case of "sunken brain syndrome,"3 downward deviation of brain parenchyma due to air collection following removal of cerebrospinal fluid caused clinical symptoms manifested by a delay in recovery from anesthesia and aggravation of focal neurological deficits. In this condition, intracranial pressure was rather low, and replacement of air with saline solution yielded restoration of brain parenchyma, with resultant dramatic clinical recovery.

I have used percutaneous subdural tapping in patients with probable tension pneumocephalus and sunken brain syndrome. This procedure was less invasive and enabled removal of air and replacement with saline at the bedside without burr-hole opening.2

The authors proposed the Mt. Fuji sign as the characteristic CT feature in tension pneumocephalus and differentiated it from a peaking sign. Judging from their CT presentations, the cardinal difference seemed to be a single peak formation in a peaking sign, and a double peak formation in the Mt. Fuji sign. However, most CT features that have been described as a peaking sign in the previous literature included double-peak formations4 Thus, although proposed mechanisms creating peaking of the frontal lobes may not be identical, it is hard to accept the Mt. Fuji sign as a separate new finding.

Finally, it is interesting to note that cases of tension pneumocephalus were occasionally complicated by remote hematoma.5 This observation may suggest that both phenomena may be secondary to common pathological processes.1

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References
RESPONSE: We appreciate the comments of Drs. Ectors, Martens, and Aoki. We agree with Dr. Aoki's opinion about the existence of "sunken brain syndrome." We would like to emphasize that there may be not only symptomatic pneumocephalus without tension ("sunken brain syndrome") but also asymptomatic pneumocephalus with tension, which was highly suspected in one of our patients in the group with asymptomatic pneumocephalus. We think that these complicated aspects of subdural pneumocephalus confuse the computerized tomography (CT) findings that have been reported in the literature. The cardinal difference between a peaking sign and Mt. Fuji sign is not the number of peaks. We agree that most CT features described as a peaking sign in the literature have included a double-peaking formation. However, the importance of air between the frontal tips has not been emphasized in tension pneumocephalus. In our opinion, it is important that the tense air widely separates the interhemispheric space between the frontal tips. It therefore creates the characteristic two peaks. As Drs. Ectors and Martens stated, arachnoidal tearing can occur at any time during the pre-, peri-, or postoperative period. Judging from our operative procedure, it must be impossible for small air bubbles to enter the cisternal subarachnoid space through an arachnoidal tear during the pre- or perioperative period. We believe that the small air bubbles enter the subarachnoid space through an arachnoidal tear postoperatively and are forced to proceed to the deep cisternal subarachnoid space by a large volume of subdural air under pressure.

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Development of Glioblastoma After Medulloblastoma

TO THE EDITOR: I read with interest the report of Schmidbauer, et al., concerning a glioblastoma that developed 6 years after a medulloblastoma (Schmidbauer M, Budka H, Bruckner R, et al: Glioblastoma developing at the site of a cerebellar medulloblastoma treated 6 years earlier. J Neurosurg 67:915-918, December, 1987). The authors contend that chemotherapy and/or radiotherapy likely played a role in the genesis of the subsequent tumor. Some questions arise concerning these assertions.

The authors cite a report by Kliriga, et al., dealing with the appearance of a glial tumor at the site of a posterior fossa medulloblastoma treated 11 years earlier. Also cited is a report by Rubinstein, et al., of a medulloblastoma that had both astrocytic and glial cell expression on tissue culture. Not mentioned by the authors are the data of Quest, et al., concerning tumor recurrence in cases of medulloblastoma. Given that the period of risk described by "Collins' law" for embryonal tumors (that is, 9 months plus the age of patient in months at presentation) was not exceeded in the case of Schmidbauer, et al., one cannot assert that the tumor that developed at the exact site of the previous medulloblastoma was unrelated.

We have described the case of a patient who developed supratentorial cerebral glioma many years after eradication of a posterior fossa medulloblastoma by means of radiotherapy. Neither computerized tomography nor cerebral arteriography revealed recurrent tumor at the site of the previous medulloblastoma. We believe that our case likely represented an instance of radiation-induced glioma. In light of the above articles, it is not certain that the same claim can be made for the case of Schmidbauer, et al.

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References

RESPONSE: Dr. Kushner rightly notes that our patient was well within the period of risk for tumor recurrence according to "Collins' law" when he developed the glioblastoma at the cerebellar site where the medulloblastoma had been treated 6 years earlier. However, this does not necessarily mean that any tumor that develops within the period of risk must be a true recurrence of the original tumor. We discussed the possibility of a medulloblastoma recurrence in detail in our paper but concluded that the new tumor phenotype was more likely a result of the applied intensive multimodality treatment, acting either by influencing tumor remnants or primitive stem cells, or by inducing a tumor de novo. Our patient's second tumor was, at least in its phenotype, clearly distinct from the original one, and thus does not fit the definition of a recurrence, which should show the same histopathology.

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Angiographically Occult Intracranial Vascular Malformations

TO THE EDITOR: Lobato, et al., should be congratulated on their extensive review of occult vascular malformations (Lobato RD, Perez C, Rivas JJ, et al: Clinical, radiological, and pathological spectrum of