Safe entry point

TO THE EDITOR: In their recent article, Hwang et al. (Hwang SC, Im SB, Kim BT, et al: Safe entry point for twist-drill craniostomy of a chronic subdural hematoma. Clinical article. J Neurosurg 112:1265–1270, June, 2009) noted that no major vessels were located within the region 1-cm anterior to the coronal suture at the level of the superior temporal line. They therefore considered that region to be a safe entry point for twist-drill craniostomy of a chronic subdural hematoma (CSDH). We encountered a rare case of a right frontoparietal CSDH associated with latent moyamoya disease (unpublished data). We chose to make a regular bur hole at the point of maximal thickness of the CSDH, at the right parietal bone, approximately 3 cm behind the coronal suture. No branches of the middle meningeal arteries were detected on the dural surface during surgery. However, a cerebral infarction in the right parietal cortex, near the bur hole, occurred after surgery. Cerebral angiography revealed total occlusion of the bilateral internal carotid arteries, with ethmoidal moyamoya vessels, leptomeningeal anastomoses, and transdural anastomoses, consistent with moyamoya disease. A branch of the middle meningeal artery passed through the margin of the bur hole. We suspected that bur-hole surgery might have caused the cerebral infarction as a result of injury to the small vessels involved in transdural anastomoses.

This case supports the suggestion of Hwang et al. regarding the safe positioning of bur-hole entry points. In cases of CSDHs, bur holes should be made at a point where no major arterial branches are located, in case the patient has latent moyamoya disease.

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RESPONSE: No response was received from the authors of the original article.

Hemangioblastoma

TO THE EDITOR: I read the case report by Utsuki et al. (Utsuki S, Oka H, Sato K, et al: Fluorescence diagnosis of tumor cells in hemangioblastoma cysts with 5-aminolevulinic acid. Case report. J Neurosurg 112:130–132, January, 2010) with interest. The authors demonstrated the first case of intraoperative fluorescence of tumor cells in the cyst wall after excision of a nodular lesion in cerebellar hemangioblastoma using 5-aminolevulinic acid (5-ALA). However, there was no mention about the fluorescence of the enhanced nodular lesion before excision. The enhanced nodular lesion of a hemangioblastoma is the core mass of the tumor cells consisting of stromal and vascular cells. Therefore, if the fluorescence-based diagnosis with 5-ALA can be consistently made in hemangioblastoma, one imagines that the nodular lesion may have shown strong fluorescence. The intraoperative fluorescence diagnosis is expected to be very useful in some cases of hemangioblastoma such as when the nodular lesion is so small and flat that the difficulty lies in finding the lesion on MR images as well as under microscopic investigation. Unfortunately, this was not our experience.

A 65-year-old man presented with gradual aggravation of dizziness, nausea, and dysarthria. At another hospital, a large cystic lesion in the cerebellar vermis had been previously diagnosed and the patient had undergone 3 surgeries in 3 years under the impression that the lesion was a hemangioblastoma. The nodular lesion around the cyst was barely visible on the previous MR images, and each operation consisted of only cyst puncture because the identification of nodular lesion after craniotomy was unsuccessful. At admission the other institution’s follow-up MR images revealed a tiny, suspicious, nodular lesion at the anterior part of cyst, which could be found only on a coronal and a sagittal image. I decided to try 5-ALA for an intraoperative fluorescence diagnosis based on the aforementioned idea, as difficulty in detection of the nodule was expected. The body weight of the patient was 67 kg, and 1.35 g of 5-ALA (Giovan, Medac GmbH) was administered orally 3 hours before the induction of anesthesia. Revision midline suboccipital craniotomy and exploration under microscopy (Leica M525 OH04 with FL400 module, Leica Microsystems GmbH) were performed. Fortunately, I could find the enhanced nodular lesion with comparative ease thanks to the aid of a navigation system. However, I failed to detect red fluorescence in the nodular lesion or any other area of the cyst wall despite multilateral efforts with the microscope. The nodular lesion was totally removed, and histological examination confirmed the diagnosis of hemangioblastoma.

Based on my experience, I wonder if Utsuki and colleagues had observed any fluorescence in the nodular lesion and if they had had consistent results in subsequent cases. The disparity in the equipment or the technical pitfalls may be the reason for the conflicting results. There may be heterogeneity in the fluorescence emission among subgroups of hemangioblastoma. Whatever the reason, there is still insufficient evidence favoring the use of 5-ALA for most benign tumors. Further accrual of experience is mandatory to bring the 5-ALA application in hemangioblastoma to routine clinical practice.

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RESPONSE: Dr. Park’s letter concerning questions raised by our case report was greatly appreciated. As he pointed out, no mention was made of the enhanced nodu-
lar lesion exhibiting fluorescence in our report. However, prior to resection, the mass did strongly demonstrate fluorescence, just as Dr. Park wondered. It would be unusual for any residual tumor to do so if this were not the case.

We also agree with Dr. Park on the utility of 5-ALA for fluorescent delineation of hemangioblastoma. When completely excised, this neoplasm should not recur, but microscopic investigation (and thus eradication) may be difficult if the lesion is small or has a membranous element. Furthermore, diagnostic fluorescence, as with 5-ALA, cannot detect residual tumor at the level of a single cell. Recurrence after gross-total resection is therefore certainly feasible.

Successful implementation of 5-ALA for intraoperative diagnostic fluorescence relies on both the quantity of protoporphyrin IX (PPIX) generated and the intensity of light exposure. Protoporphyrin IX production subsequently depends on existing tumor volume, the dose of 5-ALA given, and its postadministration time interval, as well as the ability of tumor cells to yield PPIX. Accumulated PPIX in a tumor characteristically peaks 3–6 hours after 5-ALA administration, so a 20-mg/kg dose of 5-ALA should suffice. With these points, Dr. Park has no objection.

On the other hand, residual tumor volume (the quantity of PPIX) and the strength of excitation light used in Dr. Park’s case and in ours apparently differ and might be reconciled by two improvements. The first would be to strengthen the output of activating light. Not having Leica Microsystems as a reference point, we are unsure of the light output. It also seems that the excitation light source was remote (possibly 250 mm or more). The intensity of light striking tumor cells would naturally diminish as distance from the light source increases. In our experience, tumor may fail to exhibit fluorescence if the excitation light is not in close range.

We routinely use a semiconductor laser device (VLD-V1, version 2; M & M Co., Ltd.) for excitation light. With this apparatus, the target region is exposed via optical fiber to a 40–140 mW output at a peak wavelength of 405 ± 1 nm and a striking distance of probably 10.0 mm or less. Given sufficient light activation in proximity to tumor, perhaps the tumor would have exhibited fluorescence for Dr. Park.

Another suggestion is to measure a spectrum of PPIX, thereby capturing even weak fluorescent emission. There is little quantitative PPIX when the tumor volume is limited, and assessment of PPIX fluorescence is not a macroscopic function. Without spectroscopic analysis, which was lacking in Dr. Park’s case, minor tumor fluorescence may go undetected.

It has yet to be determined whether intraoperative 5-ALA–based fluorescence lowers recurrence rates in cases of hemangioblastoma by eliminating residual tumor. Although we have achieved excellent results with our particular approach for detecting low-level PPIX fluorescence, one distinct disadvantage is that a wide surgical field cannot be surveyed in continuity, in contrast to when using Leica Microsystems.

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Please include this information when citing this paper: published online January 28, 2011; DOI: 10.3171/2010.10.JNS101671.

Chronic subdural hematoma

To The Editor: Having reviewed the recent work by Miranda et al.4 (Miranda LB, Braxton E, Hobbs J, et al: Chronic subdural hematoma in the elderly: not a benign disease. Clinical article. J Neurosurg 114:72–76, January, 2011), we would like to make the following observations on the basis of our personal experience and above all since we have been quoted on a number of a occasions in the article.

First, the authors report a cohort of patients with relatively high age. We are unaware if this is due to the particular location of the hospital or the characteristics of the population under study, such as whether patients were from a nursing home for the elderly.

Second, the authors state: “...our population is the oldest cohort yet to be reported in the literature.” In 2001, we published our experience with 90 patients older than 80 years of age (mean 88 years) who underwent bur hole and drainage, and the mortality rate in the 1st month was 7.7%.1 It should be noted that, unlike Miranda and colleagues’ series,4 our population was selected.

Thirdly, it is highly unusual to include in the same series operated and nonoperated cases. In our service, as in other cases reported in the literature, it is an exception to hospitalize a patient with a small chronic subdural hematoma (CSDH) and good neurological status (Markwalder Grade 1), the standard procedure being outpatient follow-up.

We do not agree with the authors that the type of surgery does not influence morbidity and mortality rates. Currently, most neurosurgeon are of the opinion that 1 or 2 bur holes and deployment of a subdural drain is the gold standard for the treatment of CSDH with fewer complications and lower mortality rates reported in all the recent series.2,3,5,6

Finally, the mortality rate reported by the authors is most impressive in comparison to other series reported in the literature.5,6 The authors, however, fail to state the mortality rate of surgically treated patients as opposed to patients who did not undergo surgery. It should be noted that patients with small CSDH and good neurological status (that is, non-surgical status) may die of hematoma, possibly due to causes other than CSDH (for example, cardiac or respiratory disorders). Hence, there is the importance specifying the cause of death.