tain and there are no normal controls, and this is a potential weakness of this study. However, the main objective of this study was to observe any change in regional cerebral blood flow of patients with ADC before and after therapeutic intervention with clinical correlation, and we believe our observations to be valid. This study demonstrates preliminary evidence of the efficacy of atevirdine in the treatment of ADC.

CONCLUSION

It is important to develop therapies for the treatment of ADC because, until now, only one drug, AZT, has shown efficacy in adults. The promising results of atevirdine in this study needs further investigation. This study adds to the existing data on the usefulness of cerebral SPECT in the detection and assessment of therapeutic responses in ADC.

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Parapharyngeal Meningioma Extending from the Intracranial Space Evaluated by FDG PET

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We report a rare case of parapharyngeal meningioma extending from the intracranial space evaluated by PET with [^{18}F]-2-fluorodeoxyglucose (FDG). Although the parapharyngeal meningioma had a high rate of glucose metabolism, it was proved to be pathohistologically benign. The high rate of glucose metabolism of the tumor reflected tumor aggressiveness well because the tumor grew in a relatively short time.

Key Words: parapharyngeal meningioma; PET; fluorine-18-fluorodeoxyglucose

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CASE REPORT

A 66-yr-old woman had a history of left-sided oculomotor paralysis that had been getting worse for 6 mo. CT and MRI showed a large parapharyngeal tumor. This tumor extended from the left parasellar region to the parapharyngeal space via the foramen ovale. After administration of gadopentetate dimeglumine, the tumor was moderately enhanced. The intracranial portion of the tumor invaded the cavernous sinus and surrounded the left internal carotid artery (Fig. 1A). A left carotid angiogram showed that the intracranial portion of the tumor was fed by small branches of the internal carotid artery. The extracranial portion of the tumor was fed by branches of the left external carotid artery.

We examined the glucose metabolism of the tumor using PET with ^{18}F-fluordeoxyglucose (FDG). PET was carried out with a Headtome V PET scanner (Shimadzu, Kyoto, Japan), which provides 47 tomographic slices and an in-plane spatial resolution of 4.0 mm FWHM and an axial spatial resolution of

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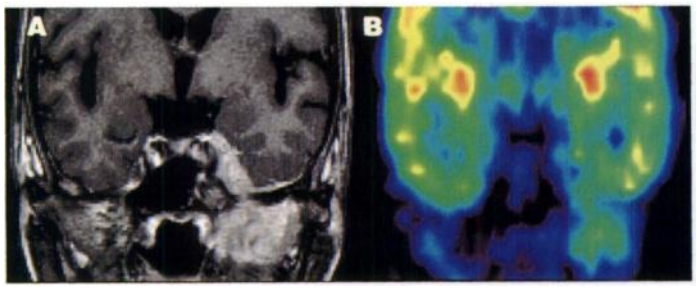

FIGURE 1. (A) Contrast-enhanced coronal T1-weighted image (gradient-recalled echo, 450/9; flip angle, 90°) shows moderately enhancing tumor in left parasellar region extending into parapharyngeal space via foramen ovale. Tumor also invades cavernous sinus and infraorbital fissure and surrounds left internal carotid artery. (B) FDG PET demonstrates that central part of extracranial tumor is as hypermetabolic as temporal gray matter.

4.3 mm FWHM (*1*). After a transmission scan was obtained, a dose of 259 MBq ^{18}F-FDG was injected into the cubital vein over 1 min. Static scanning was performed for 12 min at 60 min after the ^{18}F-FDG injection. We evaluated quantitatively the accumulation of ^{18}F-FDG using the ^{18}F-FDG method of Phelps et al. (*2*). The metabolic rate for glucose (MRGlu) was calculated by use of a fixed rate constant of 0.52 (*3*). After MRI-PET image registration was performed by the method of Ardekani et al. (*4*), we selected round regions of interest 20 mm in diameter in the tumor and normal gray matter. The MRGlu in the extracranial portion of the tumor was 4.8 ± 0.6 (mean ± s.d.) mg/100 g/min. The MRGlu in the gray matter of the temporal lobe contralateral to the tumor was 5.3 ± 0.5 mg/100 g/min (Fig. 1B).

On the basis of the results of the neuroradiological studies, including FDG PET, we diagnosed this tumor as a biologically aggressive meningioma with extracranial extension. To avoid injury to neurovascular structures in the cavernous sinus, biopsy was limited to the extracranial portion of the tumor. On the basis of the pathohistological examination, this tumor was proved to be a meningothelial meningioma. Using follow-up MRI, we calculated the doubling time of the tumor. The doubling time of the extracranial portion of the tumor was about 10 mo (Fig. 2). Although surgical resection and postoperative radiation therapy were planned on the basis of the results obtained from FDG PET, the patient declined to have the operation.

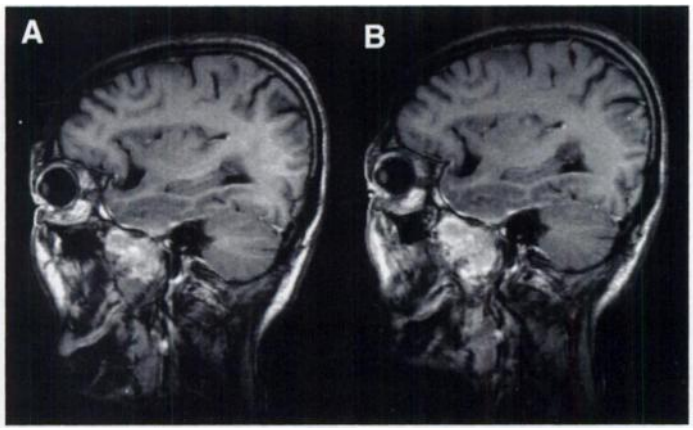

FIGURE 2. (A) Contrast-enhanced sagittal T1-weighted image (gradient-recalled echo [GRE], 450/9; flip angle, 90°) obtained before biopsy shows parapharyngeal meningioma extending from intracranial space. Tumor measured about 4 × 2.8 × 3.4 cm. (B) Contrast-enhanced sagittal T1-weighted image (GRE, 450/9; flip angle, 90°) obtained after biopsy shows growth of parapharyngeal meningioma over 7 mo. Tumor measured about 4.6 × 3.4 × 3.4 cm. Calculated doubling time of tumor was about 10 mo.

DISCUSSION

Invasive meningiomas extending into the extracranial space are rare. Friedman et al. (*5*) revealed that meningiomas in an extracranial or extraspinal site constitute fewer than 2% of all meningiomas. The mechanisms for the formation of extracranial meningiomas fall into four types (*6*). Type 1 involves direct extracranial extension from an intracranial tumor through the foramina of the base of the skull. Type 2 involves extracranial growth from the arachnoid within the cranial nerve sheaths. Type 3 involves extracranial growth from an ectopic or embryonic arachnoid cell rest without any connection to the skull base or cranial nerves. Type 4 involves distant metastases from an intracranial meningioma. Our case was thought to be classified as Type 1.

Most meningiomas with extracranial extension, such as meningothelial meningiomas, were previously reported to exhibit a pathohistologically benign appearance (*5,6*). However, according to recent reports, it is known that biological aggressiveness of extracranial meningiomas cannot be determined only by pathohistological studies. Several factors, such as invasive tendency, higher levels of proliferation, higher incidences of recurrence, multicentric growth and frequent combination with other neoplasms of the central nervous system, have been recognized as determining clinical aggressiveness in addition to pathohistologically determined malignancy (*7–9*).

Di Chiro et al. (*10,11*) reported that intracranial meningiomas could exhibit biologically aggressive behaviors without pathohistologically determined malignancy. High FDG accumulation in meningiomas was well related to biological aggressiveness. According to their studies, FDG accumulation showed a good correlation with the aggressiveness of the tumor and the clinical prognosis. Higher levels of glucose consumption correlated with more aggressive behavior of the tumor. Using a comparison with pathohistological studies, they concluded that metabolic glucose utilization rates of meningiomas were more useful than pathohistological subtypes as a method for identifying biologically active potential in the tumors.

Suwa et al. (*12*) also demonstrated invasive intracranial meningiomas without malignant pathohistological signs using a biochemical method. Using the labeling index of proliferating cell nuclear antigen, they demonstrated that meningiomas with a high labeling index of proliferating cell nuclear antigen had high proliferation (aggressiveness) potential. The tumors that they studied invaded the brain parenchyma without pathohistologically malignant transformation. Their study also supported the idea that meningiomas with biologically active potential tend to show aggressive behavior and to lead to a poor prognosis despite a pathohistologically benign appearance. Biologically active potential would be affected by complex changes on metabolic and biochemical levels more so than would pathohistologically malignant transformation.

We assessed the MRGlu of an extracranial meningioma using PET with FDG. The extracranial meningioma in this article showed high levels of glucose consumption despite a benign histologic appearance. Low rates of glucose metabolism have been shown for many meningiomas by PET with FDG (*10–14*). Compared with those for meningiomas in general (*10,11*), the doubling time of the extracranial portion of the tumor in this article was very short.

CONCLUSION

These results support the conclusion that this extracranial meningioma showed biologically aggressive behavior despite its pathohistologically benign appearance. PET with FDG is

very useful for predicting prognosis for patients with meningiomas.

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Technetium-99m-HMPAO Brain SPECT in Anorexia Nervosa

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Eating disorders have been redefined in recent years. Brain imaging techniques are useful in demonstrating the association between the morphologic and the functional cerebral changes in these cases. We report ^{99m}Tc-HMPAO brain SPECT findings in two patients with anorexia nervosa, before and after the treatment. While the detailed neurologic and laboratory examinations, including EEG and cranial CT, were within normal limits before therapy, SPECT study revealed diffuse bilateral hypoperfusion in frontal, parietal and frontotemporal areas which was more prominent in the left hemisphere. Post-treatment SPECT studies obtained after a clinical remission period of 3 mo showed normal brain perfusion in both patients. The pre- and post-treatment SPECT studies accurately reflect the functional state of the patients, and this technique may be used to follow-up the effect of treatment and predict the clinical response to therapy in patients with eating disorders.

Key Words: technetium-99m; HMPAO brain SPECT; computerized tomography; anorexia nervosa

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Anorexia nervosa is a behavioral disorder characterized by refusal to maintain body weight at or above a minimally normal weight for age and height. There is a significant disturbance in the individual's perception of the shape or size of the body and denial of the seriousness of the current low body weight (*1*).

There is an increased interest in the functional and structural cerebral changes in psychiatric disorders in the last decade. Recent studies with brain imaging techniques not only investigate the basic pathological condition in a specified disease, but also help to identify the activation phases and to discriminate the different clinical states, such as remission and exacerbation of the same disease process (*2*).

The limited number of brain imaging studies in anorexia nervosa failed to give uniform results, the main pathological finding being the cerebral cortical atrophy detected in some patients using CT (*3*). MRI studies revealed a smaller cross-sectional area of the pituitary gland and thalamus (*4,5*). An early PET study in anorectic patients revealed normal cortical glucose metabolism (*6*), while another study showed increased metabolism of the caudate nucleus in anorexic state returning to normal levels after realimentation (*7*). In a recent PET study, the underweight anorectic group showed a global hypometabolism and an absolute, as well as relative, hypometabolism of glucose in cortical regions most marked in the frontal and parietal cortices as compared to controls (*8*).

These confusing results obtained by the neuroimaging studies are mainly due to methodological discrepancies and patient selection criteria. Moreover, evaluating the patients in remission will probably lead to a better understanding of eating disorders. In this study, we investigated cerebral blood flow using ^{99m}Tc-HMPAO brain SPECT of two patients with anorexia nervosa, both at the time of diagnosis and after remission of symptoms.

CASE REPORTS

Patient 1

A 16-yr-old woman presented with a 14-mo history of intentional loss of weight (17% of the initial weight), refusal of eating because of morbid fear of weight gain, and amenorrhea for the previous 8 mo. She also had provocative vomiting.

During the last year of her college education as a successful student, although not overweight, she began dieting with the intention of losing weight. Initially, the weight loss was not recognized by the family. Gradually, she started to drop out of classes, while spending most of her time at home. Ten months after the onset of symptoms, obvious weight loss as well as the emergence of binge and purge cycles alarmed the family and she was brought for treatment.

During the initial visits, the psychiatric evaluation of the patient revealed defective self-regulatory functions, regressive pseudo-

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