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MR Imaging of Blood Flows in the Cavernous Sinus

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To evaluate blood flows in the cavernous sinus (CS), seven normal CSs and a postmortem CS were evaluated using T_1-weighted spin echo sequences. After the noncontrast study, contrast study with Gd-DTPA was performed in all cases except the postmortem one.

In the normal CSs, arterial flows of the intracavernous carotid artery (ICA) were disclosed as no-signal areas in the noncontrast study and were not enhanced with Gd-DTPA. Most venous flows in the CS were seen as low-intensity areas in the noncontrast study and were markedly enhanced with Gd-DTPA. However, these low-intensity areas were heterogeneous in intensity and several were higher and lower in intensity compared with most venous spaces. In the postmortem case, the CS showed homogeneous low intensity due to the stasis of blood flow.

It was thought that the heterogeneous appearance of venous spaces in the normal CSs was due to flow-related phenomena. This heterogeneous pattern of signal intensity suggested the distribution of flow velocities. MR demonstrated both arterial and venous flows, and this was facilitated by Gd-DTPA. MR is a promising modality for the demonstration of blood flows in the CS.

Key words: cavernous sinus, magnetic resonance imaging, blood flow, gadolinium-DTPA

Introduction

With the advances in microneurosurgery and interventional vascular surgery, the cavernous sinus (CS) is no longer an unexplored area. Increased capability to intervene in the pathology of the CS necessitates more detailed information on it. However, the CS is such a complex anatomical structure that it is difficult to observe both the vascular and neural structures using conventional diagnostic modalities. Arterial flows are well demonstrated with cerebral angiography; however, even using cerebral angiography and orbital and/or internal jugular venography, venous flows are not always demonstrated in detail.

In MR, the signal intensity of flowing blood is increased or decreased due to flow-related phenomena.[1–3] The signal increase of slowly flowing blood is called "paradoxical enhancement," which is separated into two phenomena, "flow-related enhancement" and "even-echo rephasing." The signal decrease in rapidly flowing blood is called "high velocity signal loss."[2]

The clinical efficacy of magnetic resonance (MR) imaging in demonstrating blood flows in the CS is detailed below.

Materials and Methods

Seven normal CSs in cases of supratentorial brain tumor (3 gliomas, 2 meningiomas, 1 cavernous hemangioma, and 1 metastasis; ages 20–50 years; 3 males and 4 females) underwent MR examination. All patients were free of CS symptoms, and the

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normality of the CS was verified by cerebral angiography and X-ray computed tomography (CT).

The CS in a 25-year-old dead-on-arrival (DOA) male was also examined immediately after death. Postmortem radiological examination revealed no abnormality intracranially except for static blood flow and suggested the cause of death was heart failure. Autopsy was not performed.

The MR scanner used was a 0.5 Tesla superconductive MR system (Picker International, Vista-MR). The pulse sequence was a T_1-weighted spin echo (SE) with repetition and echo times of 400–1,200 and 30–40 msec, respectively. Image planes were orthogonal; thickness was 1.0 or 0.5 cm. The data matrix was 256^2; averaging was done once or twice. The number of slices was either one, four, or eight. Image reconstruction was performed via two-dimensional Fourier transformation. After scanning without contrast medium, gadolinium-diethylenetriamine pentaacetic and (Gd-DTPA) was administered intravenously at a dose of 0.1 mmol/kg body weight in 60 seconds in all cases except the DOA male. Scanning with contrast medium was performed immediately after the administration of Gd-DTPA or within 40 minutes thereafter. Skull X-rays, plain and enhanced CT, and cerebral angiography were performed in all the normal CS cases.

Results

A. Normal CS (Fig. 1)

1. Arterial flows

The intracavenous carotid artery (ICA) was always disclosed as a no-signal area in the noncontrast study and was not enhanced with Gd-DTPA (7/7 cases). The small arterial branches from the ICA were not disclosed in any case. The ICA on coronal images was always round-shaped and no artifact attributable to arterial pulsation was observed around it.

2. Venous flows

Most venous flows in the CS were disclosed as areas of low intensity, almost the same as the cerebral cortex in the noncontrast study; these low intensity areas were markedly enhanced with Gd-DTPA (7/7 cases). In the noncontrast study, however, venous flows were not always homogeneous: several were higher and lower in intensity compared with most venous spaces. They were usually small, but there were relatively large areas of high intensity in one case and very low intensity (but not no-signal areas such as the ICA) in two. These relatively large areas with signal intensities different from most venous spaces were round-shaped, and their diameters were almost the same as that of the ICA. Very low-intensity areas were also enhanced to some degree with Gd-DTPA, but not so markedly as were most venous spaces. In one patient who underwent repeated non-enhanced study, the intensity pattern of the venous spaces was changed: a relatively large area with very low intensity disappeared and a high signal intensity area appeared in the repeated study (Fig. 1g).

3. Cranial nerves

Cranial nerves were identified as slightly lower in intensity than most venous spaces on coronal images (3/7 cases). They were located at the lateral portion of the CSs. These cranial nerves were not enhanced with Gd-DTPA and were easily recognized in the enhanced study because the surrounding venous spaces were markedly enhanced.

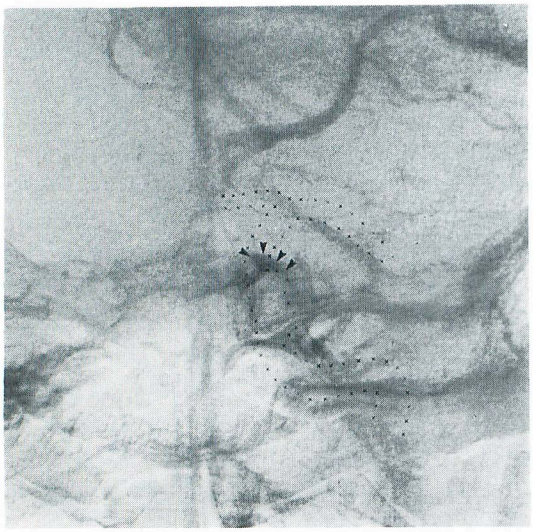
B. CS in the DOA case (Fig. 2)

The CS showed neither high nor very low signal areas and was disclosed a homogeneous area of low intensity. The ICA and venous spaces were not identified separately due to their identical signal intensities. Cranial nerves were not identified.

Discussion

The CS is different in structure from the other cerebral venous sinuses like the superior sagittal, transverse, and sigmoid sinuses. It contains the ICA, neural structures, and many influxes and effluxes of the venous channels connecting with the CS, for example, the superior and inferior opthalmic veins, sphenoparietal sinus, middle and inferior cerebral veins, superior and inferior petrosal sinuses, pterygoid veins, and anterior and posterior connections with the contralateral CS. Although the anatomy of the CS has been extensively studied by many researchers,[4–10] its venous flows are not fully understood. Most studies of the CS have been conducted on cadavers, using microscopic techniques. Cadaver studies make it difficult to recognize venous spaces because they are often collapsed and filled with clots. Venous flows in the CS can be demonstrated in several ways. The venous phase of cerebral angiography and venography (orbital and internal jugular) do not always demonstrate them in detail. Although enhanced CT also demonstrates the CS and the low-density structures representing the cranial nerves,[11] it cannot provide anatomical information as to venous spaces. Dynamic CT is necessary for demonstrating the ICA.[12]

There are two basic flow-related phenomena in



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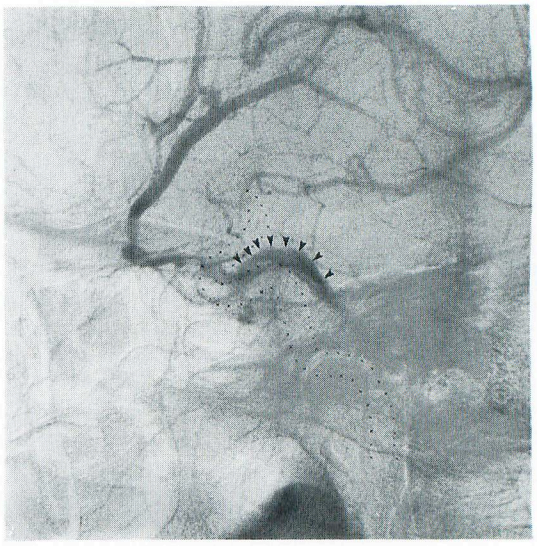

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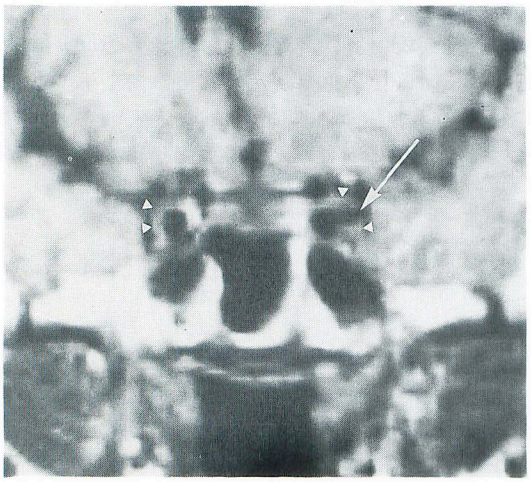

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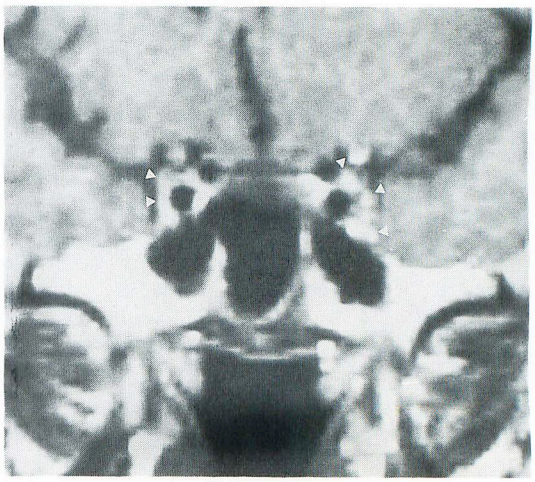

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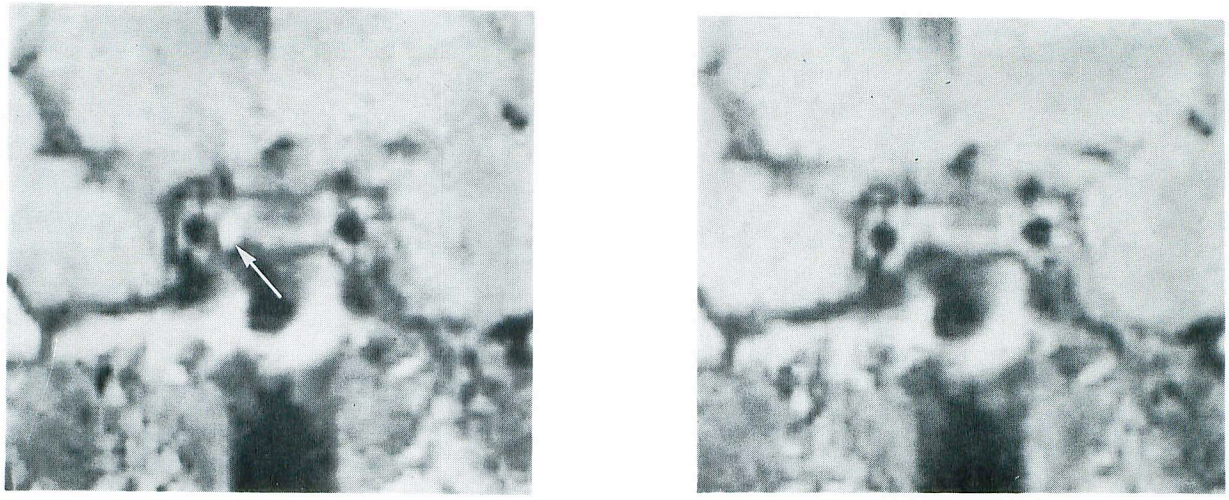

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Fig. 1. Normal cavernous sinus. a: and b: Venous phase of left carotid arteriograms. *Dots* indicate the superimposed internal carotid artery; *arrowheads* indicate partially visualized venous flows in the cavernous sinus (CS). c: T_1-weighted spin echo (SE) image without Gd-DTPA. Repetition time (msec), echo time (msec), and slice thickness (cm) area as follows: SE 500/40/0.5. Venous spaces show heterogeneous appearance, but most show low signal intensity (*arrowheads*). Note very low intensity area (*arrow*) abutting the left intracavernous carotid artery (ICA), which is thought to be high velocity venous flow. d: Enhanced T_1-weighted SE image with Gd-DTPA. SE 500/40/0.5. Gd-DTPA enhances venous spaces of the CS (*arrowheads*). This increase in signal intensity is attributable to proton relaxation enhancement of gadolinium ions (Gd^{3+}). Although the ICA remains as a no-signal area, the very low intensity area shown on image c has increased its intensity to some extent. e: T_1-weighted SE image without Gd-DTPA, 1.0 cm lateral to mid-sagittal plane, SE 600/40/0.5. The ICA is disclosed as a no-signal area; the surrounding CS shows low signal intensity. f: Enhanced T_1-weighted SE image with Gd-DTPA, the same plane as image e, SE 600/40/0.5. Although the ICA remains a no-signal area, the CS shows marked high intensity. g and h: nonenhanced SE images of the same plane, imaged with different pulse sequences. g: SE 800/40/0.5. h: SE 1,200/40/0.5. These single scans were obtained on a different day than images c-f. Flow-related enhancement occurs maximally on image g (*arrow*). Very low intensity area noted on image c is not observed on these images. The reason for this is not clear, but it is suggested that the velocity and the size of venous flows in the CS change with time and space.

MR: (1) paradoxical enhancement (flow-related enhancement and even-echo rephasing) and (2) high velocity signal loss.[2] Paradoxical enhancement is present in slow flows, whereas high velocity signal loss occurs in rapidly flowing blood.

Although only one case was available, MR imaging of the DOA patient provided useful information, namely, that the CS without blood flows showed homogeneous signal intensity. The ICA and venous spaces had the same intensity due to the lack of blood flow, and they could not be identified separately. Thus, static blood flows in the CS produced homogeneous, low signal intensity areas.

What then causes areas of higher and lower signal intensity compared with most venous spaces in the normal CS? Arterial flows in the ICA were always demonstrated as no-signal areas due to high velocity signal loss in both noncontrast and contrast studies. Gd-DTPA did not increase the signal intensity of arterial blood in the CS due to its rapidity (high velocity signal loss). Most venous flows in the CS in the noncontrast study were demonstrated as areas of low signal intensity, almost the same as the cerebral cortex on T_1-weighted SE images, but they were not no-signal areas. Static blood flows show low signal intensity, and the intensity of flowing blood in venous spaces increases or decreases depending on velocity and scanning parameters. However, venous flows were not no-signal areas like arterial flows because their velocity was less than that of arterial flows, which produced no signal due to high velocity signal loss. These results are contrary to those of Daniels' *et al.*,[13] who stated that flowing blood in the CS produced negligible signals. In the SE sequences used in the present study, only the first echo was used for image reconstruction. Thus, even-echo rephasing never occurred. Interstingly, venous flows were usually markedly enhanced with Gd-DTPA, unlike arterial flows. This may be attributable to the proton relaxation enhancement of

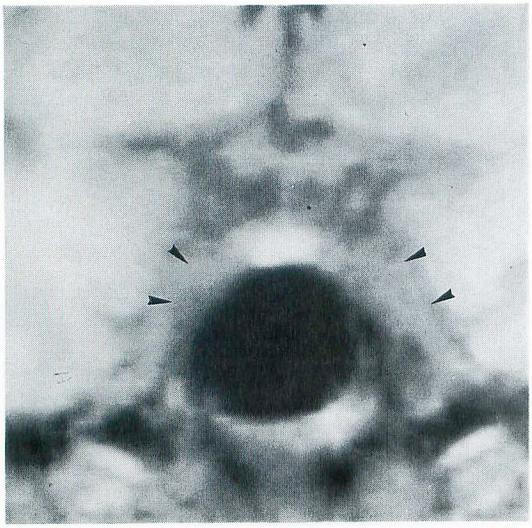

Fig. 2. Cavernous sinus of the dead-on-arrival male; cause of death was thought to be heart failure. T_1-weighted SE image (SE 600/40/0.5) was obtained immediately after death. Note homogeneous appearance of the CS (*arrowheads*). No flow-related phenomena occur due to stasis of blood flows. Because static blood flows show low signal intensity, the ICA and venous spaces show the same signal intensities.

gadolinium ions (Gd^{3+}), which produces a local magnetic field and shortens the relaxation times (T_1 and T_2) of neighboring protons.[14,15] Gd-DTPA was useful for the identification of venous spaces because enhanced areas in the CS could be identified as venous spaces, not neural structures or fat tissues.

It is possible that the areas with higher intensity than most venous spaces represented either slowly flowing blood enhanced due to flow-related enhancement, or fat tissues. Fat tissue has relatively short T_1 and T_2 values and shows high intensity on T_1-weighted SE images. Furthermore, fat images shift in the direction of the lower frequency-encoding gradient due to "chemical shift" even at an intermediate field when images are reconstructed via two-dimensional Fourier transformation. While it is possible that some of them were fat tissues, it is thought that these areas of higher intensity were attributable to slowly flowing blood, the velocity of which yielded a higher intensity than that of the other venous spaces when scanned with the given scanning parameters, because CT revealed no fat tissue and no chemical shift could be identified in the present cases.

It is possible that the areas with lower intensity than most venous spaces were representative of rapidly flowing venous blood, cranial nerves, turbulent flow, or turbulence cased by transmitted pulsation from the adjacent ICA. Cranial nerves were easily identified because they were small, round-shaped, and located at the lateral portion of the CS; Gd-DTPA did not enhance these structures. The complicated structure of the CS and its many influxes and effluxes can create turbulence in venous flows, causing loss of intensity due to the increased random motion of protons. The pulsation of the ICA can also cause turbulence of venous flows around the ICA. Because these areas did not always abut or surround the ICA, it is not likely that they were produced by transmitted pulsation from the ICA. Further, because the shapes of the relatively large areas with very low intensity seen on coronal images in two cases were almost round, it is not likely that they were caused by turbulent flow. Although it remains a possibility that some of them were caused by turbulence, it is thought that areas with lower intensity than most venous spaces were due to rapidly flowing blood, except for the laterally located cranial nerves. Gd-DTPA enhanced this rapidly flowing blood less than most venous spaces. It was thought that the velocity of these venous flows was less than that of arterial flows and greater than that of most venous flows.

Thus, the heterogeneous intensity pattern of venous spaces of the CS suggested the velocity distribution of venous flows. Furthermore, there were various sizes of venous flows in the CS. These findings support the idea that the CS is a plexus of many veins.[4] As illustrated in Fig. 1, the intensity pattern of venous spaces in the CS changed in repeated studies of the same case. The reason for this is uncertain, but it is assumed that the velocity and size of the venous channels change with time and space.

Although we did not perform subquantitative and directional analyses of blood flows, MR was able to provide information on both arterial and venous flow dynamics in the CS, as well as on anatomical structures. This has not been the case with conventional diagnostic modalities. Our preliminary experience suggests that MR is a promising modality for evaluation of the CS.

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