Intracranial lipoma is an uncommon lesion that has been well described in both the neurosurgical and neuroradiological literature for many years. This lesion is usually only an incidental finding, but it may be symptomatic. The authors describe a case of symptomatic intracranial lipoma of the superior medullary velum with emphasis on the correlation between computerized tomography and magnetic resonance imaging in evaluation of the lesion.

**KEY WORDS** • magnetic resonance imaging • computerized tomography • intracranial lipoma • superior medullary velum

**Case Report**

This 63-year-old left-handed man presented to our clinic with complaints of 2 years' duration of increasing headaches, blurred vision, and behavioral changes, the latter noted by his wife. One year before admission his headaches became constant in nature. He denied experiencing ataxia, diplopia, sphincter incontinence, nausea and/or vomiting, or seizures. Neurological examination failed to detect any signs of Parinaud's syndrome, and funduscopic examination revealed flat ophthalmic discs. Neither ataxia nor signs of cerebellar dysfunction were noted, and the remainder of the neurological examination was entirely normal.

Computerized tomography scanning performed with a General Electric CT/T 8800 scanner,* with and without contrast enhancement, showed a large lesion in the region of the superior medullary velum. The lesion did not enhance and had an attenuation value of \( -85 \) respectively. We report a case in which a lipoma attached to the superior medullary velum, causing symptoms of noncommunicating hydrocephalus, was documented by both CT and MRI.

LipoMA involving the brain is an uncommon lesion that is usually asymptomatic. Before computerized tomography (CT) scanning became available, lipomas were most often discovered during routine postmortem examination. Intracranial lipomas are most commonly located in the rostrum of the corpus callosum as first described by von Rokitansky, and are often associated with concomitant dysgenesis of this commissure. In 1935, Krainer identified other intracranial areas where this lesion may be found, including the cisterna ambiens region, chiasmatic cistern, and Sylvian and cerebellopontine angle cisterns. However, about 25% to 30% of intracranial lipomas are located in the corpus callosum. Lipoma of the quadrigeminal plate cistern causing symptoms of hydrocephalus is even rarer, and only a handful of cases have previously been presented in the literature. In addition, lipoma of the superior medullary velum has also been described.

Although CT scanning has been of great assistance in the premorbid diagnosis of these lesions, the recent development of magnetic resonance imaging (MRI) has allowed a more precise delineation of the mass and more definitive tissue characterization. In 1985, Kean, et al., described two cases of central nervous system lipoma demonstrated by MRI; these lesions were located in the upper cervical cord and corpus callosum, respectively.

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* Scanner, Model CT/T 8800, manufactured by General Electric Co., Medical Systems Division, Milwaukee, Wisconsin.
FIG. 1. Enhanced computerized tomography scan, axial view, demonstrating a large low-density lesion (arrowhead) superimposed over the superior cerebellar vermis. The lesion has an attenuation coefficient of \(-85\) Hounsfield units (HU), compatible with fat, and is relatively homogeneous as indicated by a standard deviation of 21 HU. A moderate degree of obstructive hydrocephalus can be seen.

Hounsfield units (HU), compatible with fat (Fig. 1). The low-density lesion was homogeneous (standard deviation \(21\) HU), and there was associated enlargement of the lateral and third ventricles, with a small fourth ventricle and a compressed sylvian aqueduct. Magnetic resonance imaging was subsequently performed on a General Electric 1.5-Tesla Signa scanner,† utilizing both partial-saturation (\(T_r \ 600\) msec, \(T_e \ 25\) msec) and spin-echo (\(T_r \ 3000\) msec, \(T_e \ 30\) to 120 msec) techniques. The \(T_1\)-weighted sagittal slices on MRI showed a lesion of high signal intensity (short \(T_1\)) arising from the superior medullary velum just caudal to the quadrigeminal plate (Fig. 2). The lesion invaginated into the anterior superior cerebellar vermis and extended into the upper fourth ventricle with compression of the caudal portion of the aqueduct of Sylvius. The first-echo axial slices (relative \(T_2\) weighting) revealed a lesion with a high signal intensity (short \(T_2\)) which changed to low intensity (short \(T_2\)) on the late echo \(T_2\)-weighted views (Fig. 3). These MRI parameters are characteristic of fat. The homogeneity of the lesion suggested that no desquamated debris or other tissue elements was present, ruling out a diagnosis of dermoid cyst or teratoma, respectively.

Preoperatively, visual evoked potentials were obtained and were within normal limits. Since the patient was suffering from signs and symptoms of non-communicating hydrocephalus, and not from brain stem or cerebellar compression, a left ventriculoperitoneal shunt (medium-pressure Hakim valve) was inserted under general anesthesia. No attempt was made to attack the lesion directly.

Postoperatively, the patient experienced total relief of headaches for the first time in a year, with no new neurological deficits. Computerized tomography scanning showed a decrease in the size of the ventricles. Visual evoked responses were unchanged.

Discussion

Historically, intracranial lipomas have been encountered infrequently but, with the advent of the CT scanner, clinicians more often diagnose this entity during the lifetime of the patient. More recently, MRI has afforded an unprecedented view of lipomas, as well as an appreciation of the surrounding structural relationships.\(^8\)

In the differential diagnosis of this lesion, one must consider other congenital fatty tumors of maldevelopment such as dermoid cysts, epidermoid cysts, teratomas,\(^15\) myelolipomas,\(^10\) or angiolipomas.\(^13\) The CT attenuation values of fatty tissue range between \(-50\) and \(-100\) HU. However, Kazner, \textit{et al.}\(^7\) have shown that intracranial lipomas smaller than 25 to 30 mm in diameter may present with less negative attenuation (around \(-50\) HU) and an increased standard deviation due to partial volume averaging with the surrounding tissue. Kean, \textit{et al.}\(^8\) performed MRI using a low magnetic field scanner (0.008 T) in their two patients with lipomas: one with an intracranial lesion and the second with an intraspinal lesion. They used inversion recovery techniques that produced high signal intensity of the lesions, thereby indicating a short \(T_1\). Most nonfatty intracranial tumors have a long \(T_2\) with \(T_1\) values in the intermediate-to-long range.

† 1.5-Tesla Signa scanner manufactured by General Electric Co., Medical Systems Division, Milwaukee, Wisconsin.
In our case, the homogeneous low attenuation value of the lesion on CT scans, combined with the very short $T_1$ and $T_2$ values on MRI, are compatible with fatty tissue. Dermoid cysts are less homogeneous on both CT and MRI, since they usually contain hair, hair follicles, and other debris; this material makes the CT attenuation coefficients less negative and the $T_1$ value longer on MRI. Epidermoid cysts contain a great deal of keratin and usually have CT scan attenuation values similar to those of cerebrospinal fluid (CSF). Teratomas generally contain more than one tissue type and therefore would vary in both their CT attenuation and MRI relaxation parameters as well as in their homogeneity. Hamartomas have a $T_1$ similar to gray matter on MRI, with a slightly increased $T_2$ value.

Intracranial lipomas seem to originate from the leptomeninges. Most investigators have hypothesized that fat cells present in the leptomeninges or primitive pial cells can, by maldevelopmental mechanisms, cause a lipoma. The lesion may arise as an extra-axial mass in a subarachnoid cistern without infiltration of the brain and manifest itself clinically only because of its size and critical location, causing symptomatic mass effect. This mass effect may block the flow of CSF if the lesion is located in the superior medullary velum, as was true in our case.

The direct sagittal slices obtained by MRI surpassed CT scans in delineating the origin of the lipoma, the compression of the aqueduct, and the relationship to surrounding neural structures. Computerized tomography scanning could predict the correct histological diagnosis, but anatomical delineation was less specific using only axial CT slices. While sagittally reformatted CT images can be obtained, spatial resolution is poorer than in a direct sagittal view, which is more easily obtained with MRI. Calcification surrounding lipomas has been well described in the literature and has been observed on plain x-ray films of the skull. These calcified areas would have been missed on MRI but would have been apparent on the CT scans.

Until recently, most authors have agreed that major resection with a reasonably low morbidity is not feasible and that an unacceptably high mortality rate would ensue because of the incorporation of large vessels into the tumor. However, in 1979, Clarici and Heppner reported a case in which a lipoma of the corpus callosum was totally removed using the CO$_2$ laser. The only symptoms in our patient were severe headaches, blurring of vision, and behavioral change, which were all thought to be related to the noncommunicating hydrocephalus caused by compression of the caudal aqueduct of Sylvius by the lipoma. There were no signs or symptoms referable to compression or distortion of the brain stem or surrounding structures, such as might cause Parinaud's syndrome or an ataxic gait.

The clinical presentation in our patient indicated that treatment of the hydrocephalus would relieve his symptoms. The fact that he did not have Parinaud's syndrome may be explained by the location of the lesion at the level of the superior medullary velum, just caudal to the quadrigeminal plate. Furthermore, the patient's age, his history of atherosclerotic heart disease, and his significant carotid stenosis suggested a prohibitively high operative risk if a major procedure, such as an attempt to totally or partially resect the lipoma, were to be undertaken. A subtotal removal in this case might not have assured relief of the hydrocephalus. Biopsy to histologically confirm a lipoma was considered not necessary in view of the pathognomonic MRI and CT findings. Therefore, we elected to proceed with place-
ment of a ventriculoperitoneal shunt, an operation that carried a lesser risk. Complete relief of the headaches following shunting confirmed our presumptions of the mechanism of our patient's symptoms.

The characteristic appearance of fat on both the CT and MRI scans as well as the typical midline location of lipomas will usually enable the physician to diagnose this lesion correctly. Magnetic resonance imaging should provide the necessary anatomical definition as well as correct tissue characterization of this rare mass in vivo, so that other diagnostic tests, some of which carry significant risk, may be omitted.

We believe that our case represents the first published report of a superior medullary velum lipoma causing symptomatic hydrocephalus to be demonstrated by correlative CT and MRI studies.

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