

Early CT Detection of Intracranial Seeding from Medulloblastoma

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Since 1975, intracranial subarachnoid metastases of medulloblastoma have been detected in seven of 23 initial contrast-enhanced computed tomographic (CT) scans in children with proven medulloblastoma. Furthermore, four of the seven cases with subarachnoid seeding were diagnosed after the introduction of high-resolution contrast-enhanced CT. Only three cases of seeding had been detected in the previous 17 low-resolution cases studied with CT. Thus, it is quite likely that the incidence of subarachnoid metastases may be substantially more than the overall figure of 30% indicated by this series. This may have an impact on the treatment of these patients, since the frequent appearance of metastases may indicate the need for more vigorous chemotherapeutic regimens. One should be aware of the possibility of early intracranial subarachnoid seeding and that it can be demonstrated by contrast-enhanced CT. This is particularly true when using high-resolution scanners in conjunction with 5 mm sections through the posterior fossa.

At the Childrens Hospital of Los Angeles, medulloblastoma is the most common posterior fossa brain tumor [1]. Subarachnoid metastatic spread (particularly to the spine) from primary medulloblastoma has been described often in the literature. Our report demonstrates that the early occurrence of subarachnoid spread intracranially could be detected with contrast-enhanced computed tomography (CT) in 30% of our cases. Furthermore, the introduction of high-resolution CT may lead to demonstration of subarachnoid seeding in substantially more than 30% of the cases of primary medulloblastoma of childhood. Hence, the initial contrast-enhanced CT scan in these cases should be carefully scrutinized for possible subarachnoid seeding.

Materials and Methods

We retrospectively reviewed surgically proven cases of medulloblastoma at Childrens Hospital of Los Angeles between 1975 and September 1983. Since 1975, when an EMI scanner was installed at this institution, 23 cases of medulloblastoma have been diagnosed with the aid of an initial CT scan. Several other cases either had no initial CT, or the initial records could not be retrieved. The 23 cases that were available for review were scrutinized for evidence of subarachnoid seeding of tumor.

The primary criterion of metastases was subarachnoid enhancement on the postcontrast scan. An area of increased density on the precontrast scan and obliteration of the subarachnoid space were also considered manifestations of metastases. The scanning procedure recommended with high-resolution CT machines includes 5 mm slices through the posterior fossa before and after infusion of contrast material. Conray 60 was administered by drip infusion at the rate of 2 ml/kg. It should be mentioned that this protocol was not in use during the earlier phases of this retrospective survey (before the introduction of high-resolution scanning).

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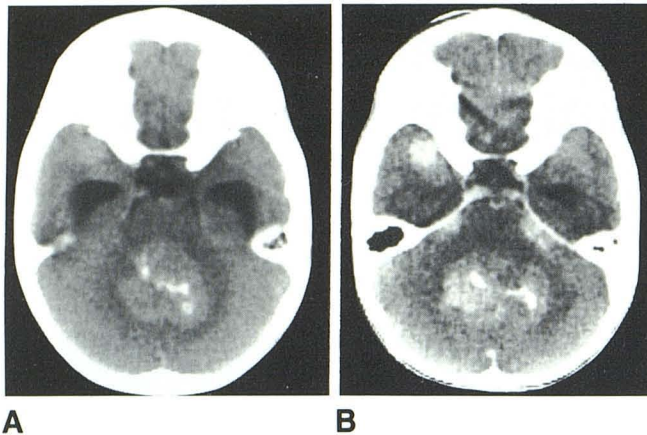


Fig. 1.—**A**, Precontrast CT scan. Faint area of nodular density in cistern of right middle cerebral artery. Centrally situated calcified primary neoplasm. **B**, After enhancement. Rather intense enhancement of nodular seeding lesion in **A**, and two more areas of nodular enhancement in cerebellopontine angle cisterns. Primary lesion exhibits enhancement also.

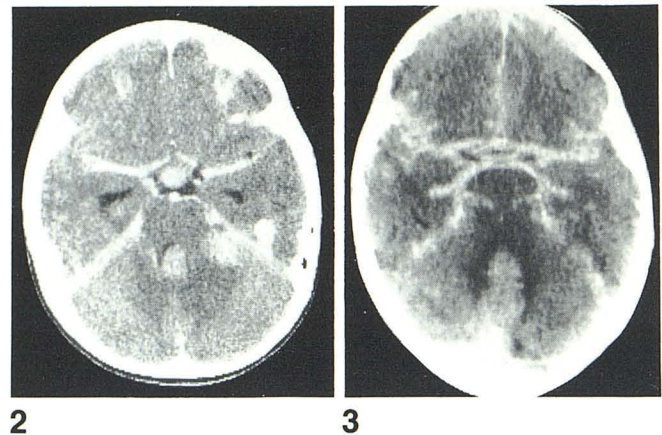


Fig. 2.—High-resolution postcontrast CT scan. Numerous areas of enhancing neoplasm. Enhancement was noted in suprasellar and perimesencephalic cisterns and cisterns for middle cerebral arteries (which were partly obliterated on noncontrast scans). Lower slices showed enhancing seeding neoplasm in angle cisterns, cerebellomedullary cistern, and vallicula. Higher slices showed abnormal enhancement in superior cerebellar cistern and upper quadrigeminal plate cistern.

Fig. 3.—High-resolution CT scan. Dramatic demonstration of sheetlike cisternal enhancement due to leptomeningeal metastatic seeding.

Results

Of the 23 cases evaluated, seven demonstrated evidence of subarachnoid seeding. The age of our patients was 18 months to 13 years (average, 5.4 years). These children were symptomatic an average of 5 weeks and usually presented with nausea, vomiting, headache, ataxia, and nystagmus. It is interesting to note that four of these seven positive cases have been found since the recent introduction of high-resolution CT scanning (GE 9800 or 8800). In fact, four of the six most recent cases of medulloblastoma that were initially diagnosed by high-resolution scanning showed evidence of subarachnoid seeding on the initial scan. All of the positive cases demonstrated abnormal enhancement in the subarachnoid space. Some showed a nodular configuration (figs. 1 and 2), but the predominant pattern was a sheetlike configuration (fig. 3). Cisternal obliteration and nodular areas of increased density were also observed in several cases. Incidentally, none of the 23 cases demonstrated evidence of ventricular metastatic lesions. Five of the seven cases with subarachnoid seeding had myelograms, and all five were positive for metastases. Only one of seven cases had positive cerebrospinal fluid (CSF) cytology.

Discussion

Subarachnoid metastases from medulloblastoma have been a popular subject in the radiologic and pathologic literature for a long time [2]. Few studies have been conducted to detect the early appearance of metastases. However, two cases reported recently with subarachnoid tumor seeding on initial CT scans [1, 3] stimulated our interest in the subject.

Medulloblastoma is reportedly the most common childhood brain tumor to show evidence of subarachnoid spread. The incidence of metastases in earlier series was 30%–50%, even

in patients with no clinical symptoms [2]. These percentages were not necessarily calculated at the time of the initial diagnosis. Subarachnoid spread of tumor occurred in 48% of the cases with CSF examination reported by Binger et al. [4]. However, these samples were also not necessarily obtained at the time of initial diagnosis. Incidentally, other researchers indicated positive CSF cytology in about 40% of the cases, but these samples generally were not obtained before tumor manipulation [5]. A recent series of Tomita and McLone [6] demonstrated five of five positive samples obtained at the time of surgery, but before tumor manipulation. This supports the thesis that the rate of seeding may be higher than the 30% detected in our series.

The occurrence of metastases has been attributed to poor encapsulation of the primary tumor, local invasion of the fourth ventricle and/or extension into the subarachnoid space, the primitive nature of the tumor cells themselves [2], as well as the viability of the tumor cells in CSF [7].

In the past, metastases to the spinal subarachnoid space have been responsible for over 90% of all of demonstrated metastases. McFarland et al. [8], in a study before CT, attributed this to the direction of CSF flow and gravitational effects.

Clark and Huettner [9] also suggested CSF flow direction as a possible explanation for the location of spinal metastases. In 1962 Stranz [10] suggested that elevated CSF pressure may be related to seeding. He also noted the contradiction that while ependymomas are more friable they have a lower incidence of seeding than do medulloblastomas. The possibility exists that very early occurrence of metastases in some instances might represent simultaneous occurrence of the tumor at multiple sites. As previously mentioned, Tomita and McLone [6] also demonstrated the probability of early subarachnoid seeding by CSF sampling before manipulation of the tumor at surgery. Because of the probability of early

metastases, treatment of the entire neuraxis has been recommended for a number of years [11, 12].

Stanley et al. [13] noted that tumor debris released at the time of surgery may be responsible for seeding, but they also noted spinal metastases on myelograms obtained at or near the time of initial surgery in three of 16 cases. The latter tends to corroborate the concept that subarachnoid seeding occurs very early in the disease process.

In another series [14], about 83% of medulloblastoma metastases appeared in the subarachnoid space. The other 17% were found in the ventricular system and were frequently accompanied by hydrocephalus. The scans in that series, however, were not obtained at the time of initial diagnosis. This probably explains the presence of intraventricular metastases in their patients, in contradistinction to our series where none of the initial scans demonstrated intraventricular metastases.

For some time now, contrast enhancement of the subarachnoid space has been known to be an indication of metastases [15]. Obliteration of the quadrigeminal cistern has also been noted as a sign of metastases, but this finding is unlikely to be reliable in the presence of an obstructing posterior fossa mass. Contrast enhancement of the subarachnoid space and obliteration of cisterns also were considered signs of subarachnoid seeding by Clark and Huettner [9], who also observed progressive increase in ventricular size in such patients. Contrast enhancement in these metastases is not surprising since the primary tumor is known to enhance very frequently [16]. However, it should be pointed out that contrast enhancement of the subarachnoid space is a nonspecific finding that can also be demonstrated in other metastatic tumors, inflammatory disease of the meninges [17], and after subarachnoid hemorrhage [18].

Our abnormal findings consisted primarily of sheets of contrast enhancement in the subarachnoid space; however, in three of the cases, small nodular masses could be demonstrated in the cisterns. It is interesting to note the strongly positive correlation between initial CT and myelography. Five of the seven cases that showed subarachnoid seeding also had myelography. Myelograms in all five cases were abnormal. The low incidence of positive CSF samples (one of seven cases) is somewhat surprising, but might be explained by the temporal relation of sampling, or the small sample volumes. In our cases less than 1 ml of CSF was generally obtained at the time of myelography.

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