Magnetic resonance imaging of pineal tumors and drop metastases: a review approach

Aikaterini G. Solomou

Department of Radiology, Medical School University of Patras, Rion, Greece

Abstract

Pineal region tumors represent less than 1% and 3-8% of brain tumors in adults and children respectively. There is a wide range of pineal masses, with the majority being germ cell and pineal parenchymal tumors. Magnetic resonance imaging (MRI) is the modality of choice for the assessment of pineal masses. It is considered as the gold standard for the evaluation of the central nervous system. MRI has the ability to produce very detailed images of the brain anatomy and is used to distinguish true pineal masses from parapineal with invasion of the gland. Specific MRI findings are helpful to the differential diagnosis of pineal tumors and the distinction between benign from malignant tumors. Pineal neoplasms may seed the subarachnoid space resulting in the development of intradural extramedullary metastases, known as drop metastases. MRI is the most sensitive method for the assessment of the spinal cord, meninges and nerve roots and the differentiation of the spinal lesions into intra/extra medullary and extradural. Because of its high sensitivity and the advances of the method, drop metastases can be easily diagnosed at an earlier stage than in the past, contributing to the selection of the appropriate treatment. Therefore, the entire neuroaxis should be investigated with MRI for the presence of intradural extramedullary lesions. The present study focuses on the main MR imaging characteristics of pineal masses and drop metastases with reference to the differential diagnosis. There is also a detailed approach to the MR protocol which should be obtained in order to evaluate the lesions.

Introduction

Pineal region tumors are rare compared to other brain neoplasms. They constitute less than 1% and 3-8% of brain tumors in adults and children respectively.[1-3] Magnetic resonance imaging (MRI) is the modality of choice for the assessment of pineal masses. It is considered to be the gold standard for the evaluation of the cen-

tral nervous system (CNS). MRI has the ability to visualize anatomical details of the brain and separate true pineal masses from parapineal with invasion of the gland. Specific MRI findings are helpful in the differential diagnosis of pineal tumors and the distinction between benign from malignant tumors. Pineal region tumors can spread through the subarachnoid space into the spinal canal resulting in the development of intradural extramedullary metastases, also known as drop metastases. MRI is the diagnostic method of choice for the detection of spinal metastases and plays a critical role in the therapeutic approach of these patients.

The present study focuses on the main MR imaging features of pineal masses and drop metastases with reference to the differential diagnosis. There is also detailed approach to the MR protocol which should be obtained in order to evaluate the lesions. Computed tomography (CT) also plays an important role and often constitutes the first imaging method but its value is decreased specifically due to the radiation and the reduced resolution compared to MRI.

Normal anatomy of pineal region

The pineal gland is a small (5-8 mm AP diameter), pine-cone shaped, midline brain structure. It is located in the quadrigeminal cistern posteriorly to the third ventricle, inferiorly to the splenium of the corpus callosum and the internal cerebral veins and superiorly to the tectal plate of midbrain. The presence of calcifications with a diameter greater than 1 cm or before the age of 10 is mainly pathologic. The choroidal branches of the posterior cerebral artery are the feeding vessels of the gland.

Pineal gland tumors

There is a wide range of pineal region masses due to the multiple different cell types found there. Tumors arising from the pineal gland are mainly classified into germ cells and pineal parenchymal tumors.

Germ cell tumors are the most common, representing more than 50% of pineal tumors.[4] They are divided into non-germinomatous and germinomatous. Based on the literature, germ cell tumors affecting the pineal region most frequently occur in males, especially non-germinomatous tumors with an incidence of 90%.[5,6]

Germinomas constitute the majority, accounting for 60-80% of all germ cell intracranial neoplasms, 3-5% of intracranial tumors in children and 0.4-1% of intracra-

nial tumors in adults.[5]

Non-germinomatous germ cell tumors (teratomas, embryonal carcinomas, choriocarcinomas and yolk sac carcinomas) are rare. Teratomas are the second more common germ cell tumor in the pineal gland.[3]

Tumors arising from pineal parenchyma account for 14-27% of pineal gland tumors.[7] Pineocytoma, pineoblastoma, pineal parenchymal tumor of intermediate differentiation (PPTID) and papillary tumor of the pineal region (PTPR) are included. Pineocytomas are reported to account for up to 30% of pineal tumors. Pineoblastoma is a common malignant tumor detected to the pineal region, with an incidence up to 50%. PPTID are less often, but account for at least 20% of this category of tumor.[8]

The remaining 25% of pineal region lesions include pineal cysts masses expanding from adjacent anatomical structures, such as gliomas arising from the adjacent brain parenchyma and meningiomas arising from the tentorium. Lipomas, pineal cysts, metastases and arachnoid cysts are also included.

Germinomas are mainly seen in children and young adults, with a peak at the age of 10-12. Approximately, 90% of the patients are less than 20 years old at the time of diagnosis.[9] Germinoma is histologically similar to dysgerminoma of the ovary and seminoma of the testis.[10] It is identified in the pineal region (50-65%), the suprasellar region (30%) and the basal ganglia-thal-

amus (5-10%).[11] Germinomas are mainly aggressive tumors that often infiltrate the adjacent anatomical structures and spread through the cerebrospinal fluid (CSF) into the spinal canal.

Teratomas are distinguished in mature, immature and those with malignant transformation, with Alpha fetoprotein (AFP) and Human chorionic gonadotropin (HCG) elevated in immature type. Teratomas more often occur in children under 10 years of age. The most common location is the pineal region, but they may also occur in the third ventricle and posterior fossa. The rest of the non-germinomatous germ cell tumors are highly malignant. AFP and HCG in CSF are elevated in yolk sac carcinoma and choriocarcinoma respectively.[7]

Pineocytomas (WHO 2016, grade I) are benign tumors, most commonly presented in adults with a mean age of 38 years old.[7] It is extremely rare for them to metastasize and spread into the spinal canal.[9,12]

Pineoblastomas (WHO 2016, grade IV) are highly malignant tumors mainly affecting children and young adults under 20 years old. The tumor infiltrates adjacent anatomical structures and often spreads into the spinal canal.

PPTID (WHO 2016, grade II or III) has histological features between those of pineocytoma and pineoblastoma without an age restriction, although there is a peak age in young adults. It rarely causes drop metastases.[9]

PTPR (WHO 2016, grade II or III) is a neuro-epithelial neoplasm arising from ependymocytes of the subcommissular organ. It affects a wide range of ages from 5 to 65 years old. Drop metastases arise in 7% of the cases.[13]

Clinical presentation

The symptoms are related to tumor local invasion and/or compression of adjacent structures, depending on the size and aggressiveness of the mass.

Compression of the midbrain anteriorly with obstruction of the aqueduct of Sylvius leads to obstructive hydrocephalus and symptoms of increased intracranial pressure. Compression of the tectal plate causes visual problems (Parinaud syndrome).[11] Infiltration of the thalamus may lead to loss of sensation and weakness on one side of the body. When the tumor expands into the suprasellar region, symptoms related to the hypothalamus may occur. Precocious puberty is a result of elevated HCG.

Imaging

CT and MRI are critical for the evaluation of the location, size and shape of the pineal tumor. The imaging findings are not specific.[14] The differential diagnosis is difficult and often a biopsy is required to determine the histological type. However, there are crucial signs that could narrow the differential diagnosis. In the presence of calcifications, the pattern of involvement is helpful. Germinomas tend to engulf the calcifications, in contrast to pineal parenchymal tumors, where calcifications are distributed. Additionally, some tumors, like pineoblastomas, infiltrate adjacent structures, the third ventricle and the basal cisterns, whereas others like germinoma exhibit edema.

A CT scan of the brain constitutes the first imaging method and can be beneficial for the assessment of the pineal region and the presence of hydrocephalus, calcifications and hemorrhage, although it should be avoided in children due to radiation.

MRI of the brain is considered the gold standard method for the evaluation of the CNS. This method is superior to CT due to the absence of radiation and its high sensitivity, the ability to illustrate anatomical details of the brain and the excellent separation of gray and white matter. MRI improves the distinction between benign from malignant tumors and the differentiation of true pineal masses from parapineal with invasion to the gland. The routine protocol consists of T1, T2, Fluid-attenuated inversion recovery (FLAIR) and Diffusion-weighted imaging (DWI) in addition to Susceptibility-weighted imaging (SWI)/Gradient echo sequences (GRE) for the detection of calcifications and hemorrhage. The use of gadolinium (Gd) in axial, coronal and sagittal planes provides information valuable to the treatment planning and enables the evaluation of how well-defined the mass is. MRI determines the site of the origin, the presence of calcifications, hemorrhage, fatty tissue or cystic components and the enhancement of the lesion, features that are crucial for the differential diagnosis. Regarding the differentiation of pineal and parapineal masses, the internal cerebral veins, the tectum of the midbrain, the superior aspect of the cerebellar vermis and the posterior aspect of the tentorial incisures should be examined thoroughly.

The imaging characteristics of pineal germinoma are not specific and include a well-circumscribed, ovoid or lobulated, relatively homogenous mass, that overwhelms the pineal gland. The signal on T1 and T2 WI is variable, most commonly slightly hyperintense on both sequences. They may demonstrate cystic or hemorrhagic compo-

nents and a tendency to invade the adjacent tissues, surrounded by edema. Markedly enhancement may be seen on MR, due to their cellularity, either homogenous or inhomogeneous with regard to their content (Figure 1). The differential diagnosis between germinoma and pineal parenchymal tumors is not accurate. A helpful sign could be the *engulfment* of the normal physiologic calcification, while pineal parenchymal tumors appear exploded.[15] If the imaging findings are present in a young male, the diagnosis is in favor of germinoma. Additionally, a hypointensity on T2WI may also be helpful in the differential diagnosis of a germinoma from a pineal parenchymal tumor.[16,17] Dumrongpisutiku *et al.*[12,14,18,19] reported that the signal on T1 and T2 WI does not illustrate significant differences between germinomas and pineal parenchymal tumors and that most of the pineal region tumors show heterogeneous enhancement, which is in agreement with other studies.

Teratoma is composed of all three germ cell lines presenting a heterogeneous appearance. The majority of non-germinomatous germ cell tumors contain fat, calcifications most often *clump like*, hemorrhage, teeth, hair, cystic and solid components.[20] Their signals on MR sequences are not specific and present a variable enhancement of the solid tissue. Hydrocephalus occurs frequently. Detection of a midline heterogeneous mass in a child should suggest the diagnosis of a teratoma.

The rare germ cell tumors have no specific imaging characteristics and may demonstrate imaging features similar to other germ cell or primary pineal tumors.[21] These lesions may contain hemorrhage, fat, or calcifications, resulting in T1 shortening. GRE sequences should be obtained in order to demonstrate and differentiate these elements. MRI can also be helpful in the evaluation of pineal parenchymal tumors. On MR, they are usually iso- to hypointense on T1WI, while on T2WI the appearance is variable, with the majority being iso- to hyperintense to gray matter.

Pineoblastoma, a highly aggressive tumor may contain hemorrhagic, necrotic or cystic foci. MRI can identify the tumor and the intrusion of the adjacent tissue but also the presence of leptomeningeal and subependymal metastases. These tumors are ill-defined because of their tendency to infiltrate the surrounding structures. On T1 WI, in the absence of hemorrhage, they have iso- or lower signal compared to the adjacent brain parenchyma. On T2 WI, the majority is isointense, unless a cystic component produces a high signal. These tumors exhibit restriction on DWI and

intense, inhomogeneous enhancement after Gd administration. Central necrotic elements must be differentiated from cystic by the administration of Gd, whereas the differentiation from a pineal cyst may be difficult.[22] Pineocytomas are well defined lesions with solid, enhanced components. Cystic or hemorrhagic areas can be demonstrated, thus the signal on T1 and T2 WI is variable. The well defined contours are helpful to the differential diagnosis in order to distinguish them from pineoblastomas (Figure 2).[20] A calcified pineal mass in a female is more likely to represent a pineocytoma. Regarding PTPR, although the presence of high signal on T1WI is a relatively specific finding, variable signal intensity on T1WI and hyperintensity on T2WI are presented. According to Chang et al the presence of high signal on T1WI may be related to the concentration of protein and glycoprotein content in the cystic regions and this may be a common imaging finding for papillary tumors.[23]

Intradural extramedullary metastases

Spinal metastases are classified as extradural, intradural extramedullary and intradural intramedullary lesions. The majority are extradural with an incidence of up to 95% while intradural extramedullary metastases account for up to 5-6% of spinal metastases. Intramedullary metastases are extremely rare, accounting for 0, 5-1%.[24]

Drop metastasis is a term for intradural extramedullary metastatic lesions of the spine that result from subarachnoid spread of a primary brain tumor. Those metastases are identified inside the dura but always outside the spinal cord. They arise from pineal tumors, ependymomas, medulloblastomas, primitive neuroectodermal tumors (PNET), glioblastomas multiform, anaplastic astrocytomas and oligodendrogliomas. The most frequent tumors that cause drop metastases in children are medulloblastomas, ependymomas, germinomas and pineoblastomas and less commonly choroid plexus neoplasms and teratomas. Drop metastases occur in 5% to 30% of children, either early, at the time of diagnosis of a brain tumor, or during the follow-up. Medulloblastoma is the most common source, accounting for half of the patients with drop metastases.[25] It is mainly diagnosed in children under 10 years of age, with a second smaller peak between 15 and 35 years old. Glioblastoma multiform is the second most common tumor with an incidence of 1% of cases and in 15% of all

patients with drop metastases.[25,26]

The two most common pineal tumors causing drop metastases are germinomas and pineoblastomas.

Leptomeningeal seeding has been reported in 6% to 14% of patients with germinomas and as high as 45% in all patients with pineoblastomas.[12,27,28]

Pineocytomas are rarely associated with drop metastases. CSF spreading is likely to occur more often in high-grade (36%) than in low-grade (7%) PPTID.[12,29] It has also been reported that PTPR may cause drop metastases in 7% of the cases.[13]

Pineal metastasis is uncommon, affecting 5% of patients with pineal tumors who underwent surgery. Lung cancer is the most common cause, however breast, kidney and gastrointestinal tract are also related. Leptomeningeal spreading is common and is present in 67% of patients.[30]

Neoplasms cause intradural extramedullary metastases by haematogenous spreading (extracranial tumors) and CSF seeding (intracranial tumors).[31,32]

Some brain tumors invade the leptomeninges or ependymal and seed the sub-

arachnoid space with cell dispersion through the CSF into the spinal canal. Another possible way is iatrogenic dispersion during a resection of a brain mass.[33,34]

The spread of the malignant cells through the spinal canal follows the pathway of the CSF, which runs along the dorsal side of the spinal cord.[35] As a result the lesions are mostly detected posteriorly. The nerve root sheaths, cauda equine and the fundus of the thecal sac may also be involved.[36]

Symptoms

The majority of the patients will suffer from back pain at the beginning, while others may have no symptoms at the time of diagnosis. Often pain is of a severe, burning nature.[31] Neurologic symptoms and mainly lower back or radicular pain will occur after the onset of pain. Subsequently, motor weakness, paraesthesias of lower extremities, gait disturbance and cauda equina syndrome may occur. One third of the patients will present anal sphincter disturbance.[37]
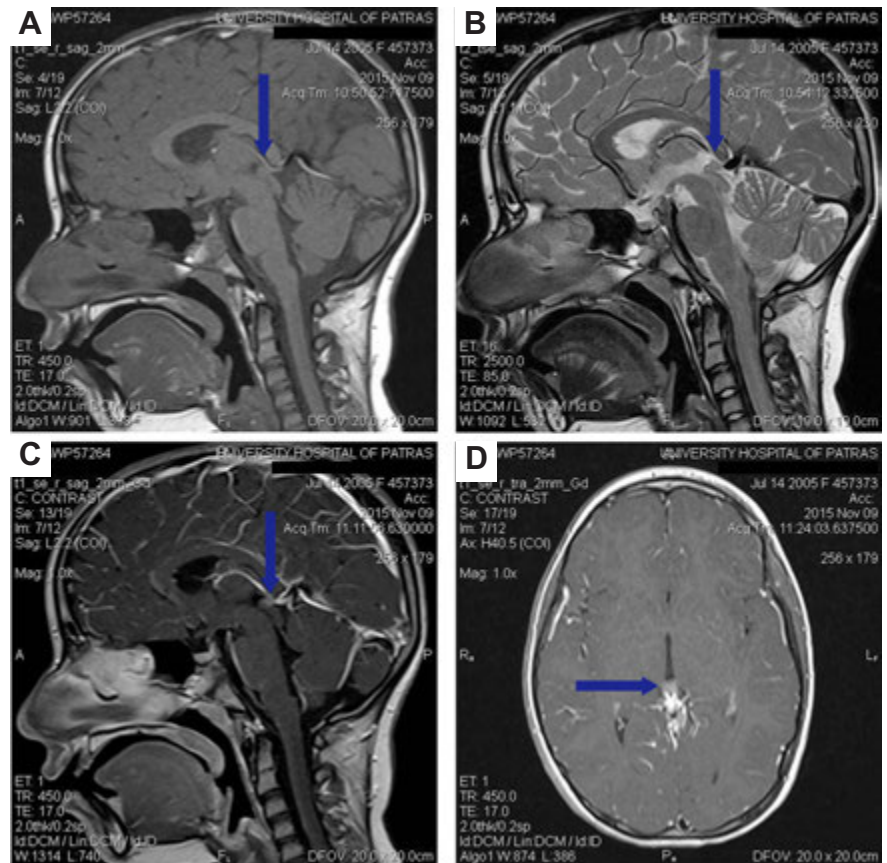

Figure 1. Pineal germinoma in an 11-year-old girl. A well - defined small pineal lesion isointense to the brain parenchyma on T1 SE (A) and T2 TSE (B) (arrow) sagittal images showing homogenous intense enhancement on post T1 SE Gd images on sagittal (C) and axial (D) planes with 2mm slice thickness (arrow).

Imaging

Drop metastases may occur at the time of diagnosis of a pineal tumor affecting surgical planning or they may occur during the treatment without recurrence of the primary tumor. Their detection is vital because survival is poor if early treatment of tumor dissemination is not performed. Drop metastases are mainly located at the lumbosacral level followed by the thoracic location but the whole neuroaxis should be investigated.

Imaging includes CT and MRI. CT is considered a poor method for the assessment of possible metastatic disease. Even though it can evaluate the spine, it may often appear normal in the pre and post contrast images.

MRI has the ability to detect drop metastases as well as to define the relationship between the lesion and the cord, to identify the presence of large feeding/draining vessels and to yield pre-operative diagnosis.[32,38-40]

The MR protocol includes T1, T2 and fat suppressed WI at least in sagittal and axial planes with Gd administration. T1WI with Gd is the most sensitive sequence, especially with fat suppression (Figure 3).

Typical imaging characteristics include nodular lesions that are isointense to the spinal cord on T1WI with intense enhancement after Gd administration. Cord edema may be present in more extensive disease, especially if there is an intramedullary component on T2 images. The detection of the nodules is more difficult on T2 WI and they are recognized by a slightly lower signal compared to CSF. T2 Fat suppressed sequences are very useful, since cord edema can be more obvious than on T2 WI.

When a solitary intradural extramedullary metastasis is detected on a nerve root, the differential diagnosis from a nerve sheath tumor is problematic.[41]

The nerve roots may show thickness and enrichment with Gd as well as the cauda equina. If drop metastases are present in the cauda equina, nodules can be detected as small isointense lesions, with considerable contrast enhancement.[42] Occasionally, diffuse enhancement may be seen in the thecal sac. In patients with leptomeningeal drop metastases or leptomeningeal carcinomatosis from non-cranial tumors, the appearance of the spinal canal mimics an effect of *sugar coating* on post-contrast images (Figure 4).[43,44] In some cases drop metastases may lead to carcinomatous meningitis or dural involvement due to inflammation, with the presence of thickened, enhanced leptomeninges. On the T1 post-Gd images, nodules and/or plaques may be identified as the tumor develops fur-
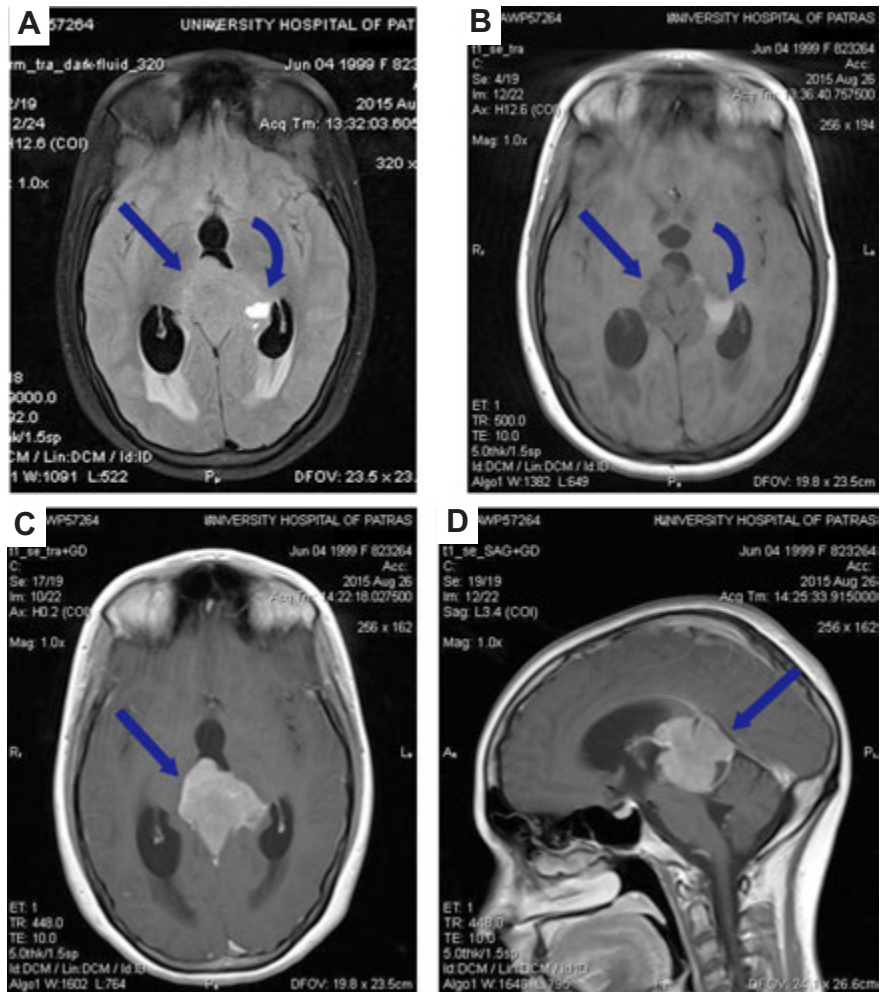

Figure 2. Pineoblastoma in a 16-year-old girl with obstructive hydrocephalus. An ill-defined inhomogeneous pineal tumor (arrow) slightly hypertensive to the adjacent parenchyma on axial FLAIR (A), hypointense on axial T1 SE (B) with hemorrhagic components (curved arrow). Axial (C) and sagittal (D) T1 SE images show the remarkable inhomogeneous enhancement of the tumor (arrow) with infiltration of the internal cerebral veins and the third ventricle resulting in obstructive hydrocephalus.
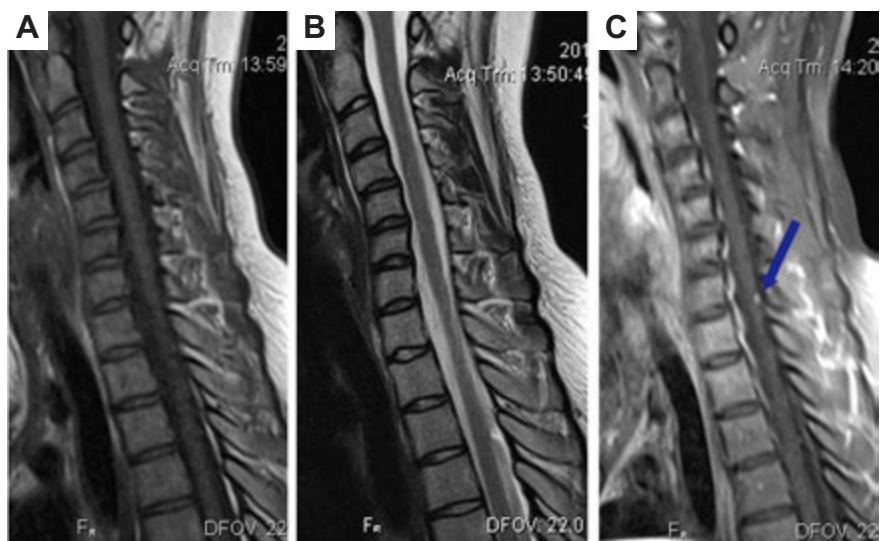

Figure 3. Solitary drop metastasis in a 16-year old girl with germinoma. On sagittal T1 TSE (A) and T2 TSE (B) the spinal canal appear normal, whereas on the post GD T1 TSE FAT SAT (C) image a solitary small enhanced lesion is present at the level of C7-Th1 posteriorly (arrow).

ther (Figure 5). In carcinomatous arachnoiditis the roots of the cauda equina appear mottled, ill-defined and sometimes they may have an asymmetric distribution within the thecal sac. The involvement of the spinal ganglion is crucial for the detection of arachnoiditis due to carcinomatosis.

Differential diagnosis

About 90% of spinal lesions that affect either the spinal canal or the vertebrae are considered to be metastatic. The most common intradural extramedullary lesions in children are drop metastases.[45,46] Extra-cranial primary neoplasms are the second source of intradural extramedullary metastases, presenting the main cause of these metastases in adults. If MRI identifies intradural extramedullary nodules with contrast enhancement then breast, lung cancer or melanoma should be excluded in adults (Figure 6). Other tumors associated are neuroblastoma, retinoblastoma and rhabdomyosarcoma.[47] Leukemia and spinal lymphoma have a high rate of leptomeningeal infiltration.[48,49]

Approximately 35% of intradural extramedullary lesions concern nerve sheath tumors including spinal schwannomas and neurofibromas which is often difficult to be distinguished because of their similar appearance.[26] They exhibit low T1 and high T2 signal with contrast enhancement and adjacent bone remodeling. Spinal schwannomas usually demonstrate more heterogeneous intensity, due to hemorrhagic and cystic components, vascular changes and fatty degeneration.[45] They may have a dumbbell shape as a result of their extradural and intradural extension through the intravertebral foramen. Neurofibromas and rarely schwannomas may present a central area of low signal on T2 that is likely to represent a collagenous stroma (target sign).[50]

Spinal meningiomas, most commonly detected in the thoracic spine, are the second most common intradural intramedullary masses in 20% of the cases. They are iso- to hypointense on T1 WI, hyperintense on T2 WI, homogenous enhanced.[51] The presence of the dural tail sign and the broad attachment of the lesion with the dura are not pathognomonic for this type of tumor.

The evaluation of the size and number of the lesions are helpful to the differential diagnosis. Nerve sheath tumors and meningiomas are larger in size than intradural extramedullary metastases.[51] Moreover, they are more often solitary and mainly occur in adults unless they are associated with neurofibromatosis (NF). Multiple
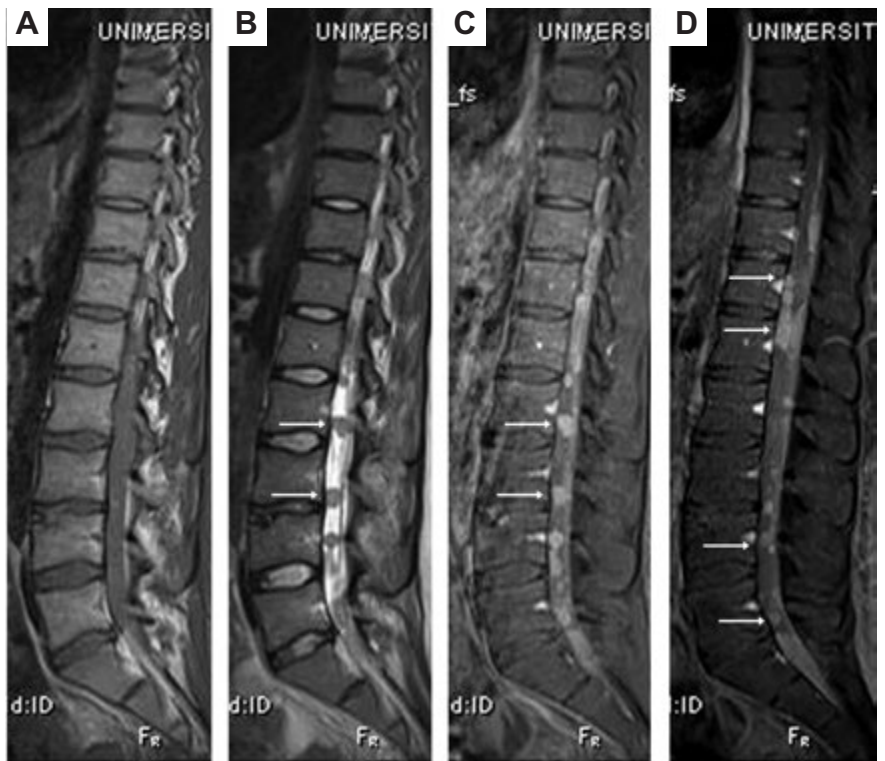

Figure 4. Multiple nodular drop metastases in the conus and the cauda equina from a germinoma. The 19 years old patient was experiencing lower extremity weakness. On sagittal T1 TSE (A) the nodules are isointense to CSF, whereas on T2 TSE (B) they exhibit low signal intensity (arrows). T1 TSE FAT SAT with Gd (C, D) illustrate markedly enhancing foci (arrows) with leptomeningeal sugar coating appearance of cord.

Figure 5. Intradural extra- intramedullary metastases from Th 10 up to the cauda eqina. On the FAT SAT Gd sequences the metastases are more obvious. On sagittal T2 TSE (A) there is an intramedullary lesion at the level Th12-L1 with intermediate high signal (arrowhead). The appearance of the spinal canal is inhomogeneous. On the post Gd images T1 TSE (B) and T1 TSE FAT SAT (C) there is an enhancement of the intramedullary lesion (arrowhead) and the dura with drop metastases (arrows).

menigiomas and schwannomas are related to NF2, whereas multiple neurofibromas correlate with NF1.

Paragangliomas are benign hypervascular tumors affecting patients between 13 and 70 years old.[52] They are rarely located in the spinal canal, more commonly to the cauda equina. They show a high signal on T2WI and contrast enhancement. Discrete flow voids may occur in this area due to the hypervascularity of the lesion as well as a rim of hemosiderin on T2 WI.[45]

Myxopapillary ependymomas represent about 50% of spinal ependymomas in adults but less than 13% in children.[53,54] On MRI the lesions show intermediate signal or less often high on T1 WI, due to mucinous component and high signal on T2 WI.[55] In the presence of hemorrhagic components, low signal on T2 WI may be present. The lesion is enhanced homogeneously depending on the extent of the hemorrhage.[55,56] Myxopapillary ependymomas may grow so much that they might expand throughout the spinal canal with scalloping of the vertebral bodies.

If there is pachymeningitis with diffuse thickening and enhancement of the meninges, carcinomatosis, infection or inflammation of the leptomeninges should be taken into consideration.

The sugar coating pattern on MRI imaging is seen in patients with intradural intramedullary metastases.

The differential diagnosis includes infectious meningitis especially in the immunocompromised patients, post infectious diseases like Guillain-Barré and inflammatory arachnoiditis often postoperatively.

Regarding inflammatory arachnoiditis there is usually a history of operation combined with specific MRI findings. Irregular thickening and clumping of nerve roots result in an empty sac sign on T2 WI, usually at the lumbar spine.

CSF laboratory examination is important for the diagnosis of infectious diseases. The clinical history of recent viral infection is typical for the Guillain-Barré syndrome. In this syndrome MRI findings are detected in the lower spine with thickening and enhancement of the conus medullaris and nerve roots of the cauda equina.

It is important not to confuse blood in the subarachnoid space postoperatively with leptomeningeal metastases. Subarachnoid or subdural blood may show high signal on T1 WI and illustrate enhancement of the leptomeninges as well. Comparison with preoperative spine MRI is useful.

Therapy

The therapeutic plan of pineal tumors depends on the histological type, the size and the presence of drop metastases.

Surgery in the pineal region is anatomically difficult and is not usually possible to completely remove the tumor. This method is usually helpful for dealing with complications like obstructive hydrocephalus and the identification of the tumor's type.

Radiotherapy and chemotherapy play an important role in the treatment of pineal tumors.[57-59]

In the presence of drop metastases, the treatment plan includes neurosurgery, radiotherapy and the grant of steroids, depend on the site and the number of the lesions. Systemic and intrathecal chemotherapy may also relieve symptoms.

Prognosis

It is extremely significant for the prognosis of patients with pineal tumors to accurately identify the presence of drop metastases. The prognosis of patients with infiltration of the neuroaxis is extremely poor and therapy is palliative with a median patient survival two to three months.

Hsieh *et al.* concluded that drop metastases found at presentation seem not to be a prognostic factor, though late drop metastases found during the follow-up reflect the relative resistance of adjuvant therapy and may be viewed as a possible poor prognostic factor.[60]

Conclusions

MRI is the gold standard method for the evaluation of pineal tumors and the detection of intradural extramedullary metastases guiding the therapeutic planning. MRI should be performed early at the diagnosis of the pineal tumors and during the follow-up providing crucial information about the stage and possible recurrence of the disease.

Figure 6. Intradural metastasis from melanoma Sagittal T1 TSE (A) and T2 TSE (B) present an oval lesion with low signal (arrowheads). On T1 TSE FAT SAT with Gd (C) the lesion shows inhomogeneous enhancement (arrow heads). A dural tail sign is illustrated (thin arrow) and although the finding is more specific for a meningioma, the infiltration of the adjacent tissues is in favor for metastasis. A biopsy confirmed the diagnosis.

References

1. Hoffman HJ, Otsubo H, Hendrick EB et al. Intracranial germ cell tumors in children. J Neurosurg 1991;74:545-51.
2. Drummond KJ, Rosenfeld JV. Pineal region tumours in childhood: a 30-year experience. Childs Nervous System 1999;15:119-26.
3. Matsutani M, Sano K, Takakura K, et al.

Primary intracranial germ cell tumors: a clinical analysis of 153 histologically verified cases. J Neurosurg 1997;86: 446-55.

4. Cheng CM, Chiang YH, Nieh S. Pineal region teratoma with high serum and CSF alpha-fetoprotein levels. J Clin Neurosci 2006;13:257-9.

5. Villano JL, Propp JM, Porter KR et al. Malignant pineal germ-cell tumors: an analysis of cases from three tumor registries. Neurooncol 2008;10:121-30.

6. Goodwin TL, Sainani K, Fisher PG. Incidence patterns of central nervous system germ cell tumors: a SEER study. J Pediatr Hematol Oncol 2009;31:541-4.

7. Louis DN, Ohgaki H, Wiestler OD et al. The 2007 WHO classification of tumours of the central nervous system. Acta Neuropathol 2007;114:97-109.

8. Osborn AG, Salzman KL, Thurnher MM, et al. The new World Health Organization classification of central nervous system tumors: what can the neuroradiologist really say? Am J Neuroradiol 2012;33:795-802.

9. Smith AB, Rushing EJ, Smirniotopoulos JG. From the archives of the AFIP: lesions of the pineal region: radiologic-pathologic correlation. Radiographics 2010;30: 2001-20.

10. Echevarría ME, Fangusaro J, Goldman S. Pediatric central nervous system germ cell tumors: a review Oncologist 2008;13:690-9.

11. Ferla S, Spart S, Giordano R, et al. Pineal germinoma: diagnosis, treatment and tumor response. Ital J Neurol Sci 1987; 8:267-70.

12. Dumrongpisutikul N, Intrapiromkul J, Yousem DM. Distinguishing between germinomas and pineal cell tumors on MR imaging. Am J Neuroradiol 2012;33:550-5.

13. Fèvre-Montange M, Hasselblatt M, Figarella-Branger D, et al. Prognosis and histopathologic features in papillary tumors of the pineal region: a retrospective multicenter study of 31 cases. J Neuropathol Exp Neurol 2006;65:1004-11.

14. Reis F, Faria AV, Zanardi VA, et al. Neuroimaging in pineal tumors. J Neuroimaging 2006;16:52-8.

15. Tony J, Westphal M, Rutka JT, Grossman SA. Neuro-Oncology of CNS Tumors. Berlin: Springer; 2006.

16. Brant WE, Helms CA. Fundamentals of diagnostic radiology. Philadelphia: Lippincott WIlliams & Wilkins; 2012.

17. Kilgore DP, Strother CM, Starshak RJ, Haughton VM. Pineal germinoma: MR imaging. Radiology 1986;158:435-8.

18. Dahiya S, Perry A. Pineal tumors. Adv Anat Pathol 2010;17:419-27.

19. Komakula S, Warmuth-Metz M, Hildenbrand P, et al. Pineal parenchymal tumor of intermediate differentiation: imaging spectrum of an unusual tumor in 11 cases. Neuroradiology 2011;53:577-84.

20. Russell DS, Rubinstein LJ. Pathology of tumors of the nervous system. 5th ed. Baltimore: Williams & Wilkins; 1989.

21. Smirniotopoulos JG, Rushing EJ, Mena H. Pineal region masses: differential diagnosis. Radiographics 1992;12:577-96.

22. Rodjan F, De Graaf P, Moll AC, et al. Brain abnormalities on MR imaging in patients with retinoblastoma. Am J Neuroradiol 2010;31:1385-9.

23. Chang AH, Fuller GN, Debnam JM, et al. MR imaging of papillary tumor of the pineal region. Am J Neuroradiol 2008;29:187-9.

24. Shah LM, Salzman KL. Imaging of spinal metastatic disease. Int J Surg Oncol 2011;2011:769753.

25. Provenzale JM, Nelson RC, Vinson EN. Duke radiology case review: imaging, differential diagnosis and discussion. 2nd ed. Philadelphia: Williams & WIlkins; 2012.

26. Choi PP, Shapera S. Drop metastases. Can Med Assoc J 2006;175:475.

27. Pacholke HD, Pincus DW, Mendenhall NP. An unusual case of pure germinoma in a patient with Tourette's syndrome. J Hong Kong Coll Radiol 2003;6:155-7.

28. Chang SM, Lillis-Hearne PK, Larson DA, et al. Pineoblastoma in adults. Neurosurgery 1995;37:383-90.

29. Fauchon F, Jouvet A, Paquis P et al. Parenchymal pineal tumors: a clinico-pathological study of 76 cases. Int J Radiat Oncol Biol Phys 2000;46:959-68.

30. Lassman AB, Bruce JN, Fetell MR. Metastases to the pineal gland. Neurology 2006;67:1303-4.

31. Perrin RG, Livingston KE, Aarabi B. Intradural extramedullary spinal metastasis. A report of 10 cases. J Neurosurg 1982;56:835-7.

32. Schick U, Marquardt G, Lorenz R. Intradural and extradural spinal metastases. Neurosurg Rev 2001; 24:1-5.

33. Norris LK, Grossman SA, Olivi A. Neoplastic meningitis followIng surgical resection of isolated cerebellar metastasis: a potentially preventable complication. J Neurooncol 1997;32: 215-23.

34. Van der Ree TC, Dippel DW, Avezaat CJ, et al. Leptomeningeal metastasis after surgical resection of brain metastases. J Neurol Neurosurg Psychiatry 1999;66:225-7.

35. Pravin S, Kirti G, Priyamvadha K. Preoperative diffuse leptomeningeal spread in a medulloblastoma: paraplegia followIng surgery for posterior fossa and call for newer management protocols. J Pediatr Neurosci 2011;6:152-4.

36. Lam CH, Cosgrove GR, Drislane FW, Sotrel A. Spinal leptomeningeal metastasis from cerebral glioblastoma. Appearance on magnetic resonance imaging. Surg Neurol 1991;35:377-80.

37. Traul DE, Shaffrey ME, Schiff D. Part I: spinal-cord neoplasms-intradural neoplasms. Lancet Oncol 2007; 8:35-45.

38. Bradley WG. Use of contrast in MR imaging of the lumbar spine. Magn Res Imaging Clin N Am 1999,7:439-57.

39. Schuknecht B, Huber P, Buller B, Nadjmi M. Spinal leptomeningeal neoplastic disease. Eur Neurol 1992; 32:11-6.

40. Zumpano BJ. Spinal intramedullary metastatic medulloblastoma. Case report. J Neurosurg 1978;48:632-5.

41. Akhaban A, Mehrabaniyan MR, Jarahi M, Navabii H. Intradural extramedullary metastasis from papillary carcinoma of thyroid. BMJ Case Rep 2012;2012:bcr0220125801.

42. Park TS, Hoffman HJ, Hendrick EB, et al. Medulloblastoma: clinical presentation and management: experience at the hospital for sick children, Toronto, 1950-1980. J Neurosurgery 1983;58: 543-52.

43. Singh SK, Agris JM, Leeds NE, Ginsberg LE. Intracranial leptomeningeal metastases: comparison of depiction at FLAIR and contrast-enhanced MR imaging. Radiology 2000;217:50-3.

44. Singh SK, Leeds NE, Ginsberg LE. MR imaging of leptomeningeal metastases: comparison of three sequences. Am J Neuroradiol 2002;23: 817-21.

45. Soderlund KA, Smith AB, Rushing EJ, Smirniotopolous JG. Radiologic-pathologic correlation of pediatric and adolescent spinal neoplasms: Part 2, Intradural extramedullary spinal neoplasms. Am J Roentgenol 2012;198:44-51.

46. Huisman TA. Pediatric tumors of the spine. Cancer Imag 2009;9:S45-8.

47. Ketonen LM, Hiwatashi A, Sidhu R, Westesson PL. Pediatric brain and spine: an atlas of MRI and spectroscopy. Berlin: Springer Verlag; 2005.

48. Yousem DM, Patrone PM, Grossman RI. Leptomeningeal metastases: MR

evaluation. J Comput Assist Tomogr 1990;14:255-61.

49. Pui MH, Langston JW, Arai Y. Gd-DTPA enhancement of CSF in meningeal carcinomatosis. J Comput Assist Tomogr 1993;17:940-4.

50. Wilne S, Walker D. Spine and spinal cord tumours in children: a diagnostic and therapeutic challenge to healthcare systems. Arch Dis Child Educ Pract Ed 2010;95:47-54.

51. Li MH, Holtas S, Larsson EM. MR imaging of intradural extramedullary tumors. Acta Radiol 1992;33:207-12.

52. Rifkinson-Mann S, Wisoff JH, Epstein F. The association of hydrocephalus with intramedullary spinal cord tumors: a series of 25 patients. Neurosurgery 1990;27:749-54.

53. Seo HS, Kim JH, Lee DH et al. Non enhancing intramedullary astrocytomas and other MR imaging features: a retrospective study and systematic review. Am J Neuroradiol 2010;31: 498-503.

54. Abel TJ, Chowdhary A, Thapa M et al. Spinal cord pilocytic astrocytoma with leptomeningeal dissemination to the brain: case report and review of the literature. J Neurosurg 2006;105: 508-14.

55. Nadkarni TD, Rekate HL. Pediatric intramedullary spinal cord tumors: critical review of the literature. Childs Nerv System J 1999;15:17-28.

56. Kothbauer KF. Neurosurgical management of intramedullary spinal cord tumors in children. Pediatr Neurosurg 2007;43:222-35.

57. Haddock MG, Schild SE, Scheithauer BW, Schomberg PJ. Radiation therapy for histologically confirmed primary central nervous system germinoma. Int J Radiat Oncol Biol Physics 1997;38:915-23.

58. Bruce JN, Kopell BH. Pineal tumors treatment and management. 2015. Available from: http://emedicine. medscape.com/article/249945-treatment

59. Balmaceda C, Heller G, Rosenblum M et al. Chemotherapy without irradiation: a novel approach for newly diagnosed CNS germ cell tumors: results of an international cooperative trial. J Clin Oncol 1996;14:2908-15.

60. Hsieh PC, Wu CT, Lin KL, et al. The clinical experience of medulloblastoma treatment and the significance of time sequence for development of leptomeningeal metastasis. Child Nerv Syst 2008;24:1463-7.