Meningeal pathology with MRI: a practical approach

Poster No.: C-1083
Congress: ECR 2013
Type: Educational Exhibit
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Keywords: MR, Neuroradiology brain
DOI: 10.1594/ecr2013/C-1083

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Learning objectives

1. To carry out an approach to the diagnose algorithm of meningeal pathology with MR, by describing the protocols and main manifestations of that pathology.

2. To show, with a practical point of view, the affected patterns in MR and to provide clues in order to make a precise radiologic report.

3. To check the cases reported in our hospital unit.
Background

I. INTRODUCTION

- Meningeal anatomy

The meninges that envelop the brain consist in three layers: the dura mater, the arachnoid mater and the pia mater. The outermost layer is the dura mater or pachymeninx which lies closest to the calvaria. Epidural space is a potential space between the outer layer of the dura mater and the inner layer of the skull. The middle element of the meninges is the arachnoid mater that is attached to the overlying dura mater with a virtual space between both layers, the subdural space. The innermost meningeal covering is the pia mater that firmly adheres to the surface of the brain. Arachnoid mater and pia mater form the leptomeninx and the space between these two layers is the subarachnoid space, which is filled with cerebrospinal fluid.

- Features normal behaviour of the meninges

The blood vessels within the dura mater lack of vessel unions, so they do not produce a blood-brain barrier in contrast to arachnoid vessels which are components of the blood-brain barrier. Because of this, normal dural enhancement is seen clearly on MR images with the following features:

- Thin, smooth, discontinuous and symmetric,
- Less strong than the enhancement of the venous sinuses,
- More prominent in the dural reflections of the falx cerebri, tentorium cerebelli and falx cerebelli.

Fig. 1 on page 11

II. IMAGING PROTOCOL or MR TECHNIQUES

Magnetic resonance (MR) is the technique of choice in the diagnosis of the meningeal pathology. MR is a very reliable tool to determine the extension of the disease and also the associated findings that could be useful to establish the differential diagnosis.

The MR imaging protocol for the depiction of meningeal disease, in addition to the brain conventional sequences, should include sequences after contrast administration because this increases the sensibility of the examination and the specificity for meningeal alterations.
Supplementary sequences in the study of meningeal lesions.

Some sequences can be performed after contrast administration besides conventional T1 images:

a) **FLAIR/T1FLAIR images.** Post-contrast FLAIR images (especially in delayed-enhanced images) have been described as a useful technique for diagnosing meningeal diseases.

b) **Fat suppression sequences** (T1 or FLAIR). In cases with osseous or soft tissues involvement and also in those cases in which the pathology is localized within a region that contains a lot of osseous structures (skull base...).

Fig. 2 on page 11

c) **T1-3D sequences** detect and define in a better way the meningeal enhancement and its features (nodular...) and also provide the opportunity of making multiplanar reconstructions. **Fig. 3 on page 12**. These sequences have some limitations because they tend to overvalue the physiological dural enhancement and, occasionally, they don´t stand out the leptomeningeal enhancement. So, it is always mandatory to perform the conventional T1 image. **Fig. 4 on page 13** **Fig. 5 on page 14**

### III. AFFECTED PATTERNS IN MR

Meningeal disease in MR can be classified in two groups: presence of enhancement or mass.

a) The "enhancing pattern": can be pachymeningeal or leptomeningeal.

- **Pachymeningeal:** dural enhancement that is adjacent to the inner table of the skull, interhemispheric fissure and tentorium cerebelli. This pattern does not draw sulci and basal cisterns and it can be diffuse or focal, lineal or nodular. **Fig. 3 on page 12**

- **Leptomeningeal:** contrast enhancement follows the subarachnoid spaces of sulci and cisterns. **Fig. 6 on page 15**

- **Please note:** It is important to keep in mind that meningeal enhancement distribution does not purely differentiate dural from leptomeningeal involvement because both layers are closely related. Therefore the presence of pachymeningeal pattern doesn´t imply that leptomeningeal covering is respected.
b) The "mass pattern": requires a detailed internal behaviour description and if there is bone or parenchymal involvement, dural tail and multiplicity of lesions.

IV. MENINGEAL PATHOLOGY

The most frequent causes of abnormal meningeal enhancement are postoperative changes, meningeal fibrosis following subarachnoid haemorrhage, infectious pathology and neoplasms.

1. Postoperative changes
2. Tumoral pathology
3. Infectious pathology
4. Inflammatory /granulomatous pathology
5. Intracranial hypotension syndrome
6. Idiopathic hypertrophy pachymeningitis
7. Miscellany

1. Postoperative changes: transient focal pachymeningeal enhancement is seen in the majority of patients undergoing a cranial surgery. This enhancement can also be diffuse, patchy and mixed (with associated leptomeningeal component). Fig. 7 on page 16

2. Tumoral pathology

a) Meningioma is the most common non-glial primary cerebral neoplasm. It can adopt "mass" shape (the more typical one) or "en plaque" shape. In the last one type there is an extensive dural thickening with sessile appearance and can infiltrate the adjacent bone. Fig. 8 on page 17

Three categories are described (WHO): typical meningioma, atypical and anaplastic. Fig. 9 on page 18

Typical meningioma features: Fig. 10 on page 19

• well defined mass,
• homogeneus signal and enhancement
• dural tail (60%): this finding suggests meningioma but it is not pathognomonic,
• calcification foci, necrosis and haemorrhage can be present as well as xanthomatos and cystic changes (10-15%). Fig. 11 on page 20
• supratentorial location (90%)
• associated peritumoral edema (50%)
• osseous destruction can be present but the most common associated osseous change is hyperostosis. Meningiomas can infiltrate adjacent bone and present extracranial component. Fig. 9 on page 18

In about 1-9% of the cases multiple meningiomas are seen (16% in postmortem studies). It is related to the antecedent of radiotherapy. Fig. 8 on page 17. Multiple meningiomas are different from meningiomatosis which is a manifestation of the type 2 neurofibromatosis.

It is important to remember that, despite meningioma can have atypical presentation, it is the most frequent meningeal tumor.

b) Other neoplasms that can originate from the meninges are: mesenchymal and osteocartilaginous tumors, primary pigmented lesions, hemangiopericytoma… Fig. 12 on page 20

Meningeal covering can be affected for a wide variety of pigmented lesions of the CNS. They can be circumscribed or diffuse and their imaging appearance depends on the amount of melanin and the presence of haemorrhage. They include:

• primary pigmented lesions of the CNS,
• metastatic melanoma, Fig. 13 on page 21
• other CNS tumors than can show melanin transformation including schwannoma, medulloblastoma and some gliomas.

Primary melanocytic neoplasms are rare lesions arising from normally occurring leptomeningeal melanocytes. They include: diffuse melanocytosis, meningeal melanomatosis (in neurocutaneous melanosis), melanocytoma and malignant melanoma. When a pigmented lesion is discovered it’s essential to look for a primary pigmented tumor outside the CNS.

The features of these neoplasms overlap because they can show diffuse leptomeningeal proliferation or little focal masses can be present. They can also have different signal intensity so frequently they cannot be distinguished from meningiomas or other neoplastic or infectious lesions.
c) **Metastatic lesions**

- **Oncohematologic diseases.** Leucemic or linfomatous cells can affect the skull diploe, the dura mater, the leptomeninges or all of them. They can have a focal or diffuse appearance. Fig. 14 on page 21

Primary leukemia of the CNS is extremely rare and it normally shows some masses with enhancement and dural base.

Secondary lymphoma usually has extraaxial location and can be epidural, subdural, subarachnoid or a combination of all of them. Fig. 15 on page 22

In these patients, infectious processes and chemical meningitis can also occur and their manifestations can be similar to the tumoral disease so the cytological exam of the cerebrospinal fluid is indispensable.

- **Cranial osseous metastases.** They are relatively common and secondarily they can infiltrate the adjacent dura mater and brain parenchyma. Fig. 2 on page 11 Fig. 16 on page 23

- **Meningeal metastases.** The most frequent tumors metastasizing to the meninges are breast and lung cancer, lymphoma-leukemia and malignant melanoma. Primary tumors of the CNS (medulloblastoma, PNET, ependymoma, germinal cells tumor, astrocytoma and glioblastoma) show affinity for the meninges.

Dural metastases have similar spectrum of presentation than meningiomas and they can also have dural tail. Fig. 17 on page 24 Fig. 18 on page 25 Fig. 19 on page 26

Meningeal carcinomatosis can affect the dura mater, leptomeninges or both. Leptomeningeal involvement is expressed as thin lines or small nodules with enhancement extending along the cortical surface. Fig. 20 on page 27

Pachymeningeal carcinomatosis is a rare type of metastatic intracraneal disease. The dura mater appears diffusely thickened as a membrane with nodular aspecto that covers the brain but not the adjacent subarachnoid space.

The presence of neoplasm near to the meninges or a small foci of meningeal tumor can produce, in addition, a diffuse fibrose response in the adjacent meninx. Both tumor and fibrosis show enhancement so the specificity of this finding is limited.
3. Infectious pathology

Infectious pathology generally produces leptomeningeal involvement. Fungal infections can show nodular thickening and enhancement, as leptomeningeal carcinomatosis does.

Some infections can associate pachymeningeal enhancement (tuberculosis, fungus...), usually with nodular appearance.

**Fig. 21** on page 28

4. Inflammatory/granulomatous pathology

Churg-Strauss syndrome, Wegener granulomatosis, reumatoid arthritis, sarcoidosis... These pathologies are associated to a characteristic clinical context. They can present CNS involvement with infiltration of meninges that produces dural enhancement with nodules and masses localized in the basal cisterns.

**Fig. 22** on page 29  **Fig. 23** on page 30

5. Intracranial hypotension syndrome

Loss of cerebrospinal fluid (CSF) pressure that can be idiopathic, after a lumbar puncture, CSF fistulae, postsurgical, posttraumatic...

The classical clinical syndrome consists in:

- Positional headache,

- Low CSF pressure,

- Pachymeningeal enhancement, usually diffuse, with possible associations (subdural effusions, prominence of venous sinuses, descent of the brain). Disappearance of these findings is confirmed in the follow up. **Fig. 24** on page 31  **Fig. 25** on page 32

Pachymeningeal enhancement after lumbar puncture without complication is normally resolved in 48-72 hours. **Fig. 3** on page 12

6. Idiopathic hypertrophy pachymeningitis
This pathology is a rare inflammatory fibrosing disease with focal or diffuse thickening of the dura mater. The most frequent manifestation is headache and it is an exclusion diagnosis.

7. Miscellany

- Radiotherapy can produce meningeal changes, normally leptomeningeal enhancement, although it can be mixed. Fig. 26 on page 33

- Cerebrovascular accident can associate leptomeningeal enhancement.

- Subarachnoid haemorrhage can be accompanied by pachymeningeal enhancement.

- Hemophagocytic syndrome is a rare childhood disease characterized by histiocytic and lymphocytic infiltration in different organs. Parenchymal alterations and leptomeningeal enhancement can be present.

- Amyloidosis can originate amilod deposits in the dura mater and simulate meningiomas.

V. DIAGNOSTIC ALGORITHM Table 1 on page 36 Table 2 on page 36

There are some points to keep in mind when elaborating the radiological report

1. CLINICAL CONTEXT

The clinical context is very important in meningeal pathology and the radiologist must pay attention to the medical history in each patient.

2. DESCRIPTION OF THE RADIOLOGICAL FINDINGS

- To make a deep and correct description of the findings
- To recognize the presentation pattern and its characteristics
- To describe the associated findings: parenchymal or osseous involvement, distribution, location and potential complications

3. CONCLUSION
It is important to delimit the differential diagnosis following the diagnostic algorithm of the meningeal pathology, with some considerations:

- When no clinical history of interest is present and pachymeningeal enhancement exits, this finding is non-specific.
- The first question to ask when pachymeningeal enhancement is present is if it has been performed a previous recent lumbar puncture.
- In the case of an extra-axial mass, whatever its features, the most frequent etiology is meningioma, taking into consideration that in an oncological context, dural metastases could show similar manifestations.
- A mass with hyperintensity in T1WI can correspond to a meningioma with previous bleeds or to a pigmented lesion.
- Leptomeningeal nodular enhancement suggests neoplastic or fungal etiology.
- Infections more frequently produce leptomeningeal pattern.
- The leptomeningeal pattern is caused more commonly by infections than tumors.
- Leptomeningeal metastases are more frequent than pachymeningeal metastases.
- Occasionally superficial tumors with large dural contact can simulate extra-axial masses. Fig. 27 on page 34
- Meningioma can also infiltrate the adjacent bone and present extra-cranial component. So in the presence of a lesion with osseous, dural and extra-cranial components the differential diagnosis will be between dural neoplasm (primary or secondary) and osseous tumor, being the last the most frequent.
- Schwannomas should be considered in the presence of a mass in the posterior fossa (ponto-cerebellar angle) or in locations where nerves are passing. Fig. 28 on page 34 Fig. 29 on page 35
Fig. 1: Axial 3D T1WI after contrast administration. Normal dural enhancement.

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Fig. 2: Axial enhanced T1WI with fat supression technique. Osseous metastases from breast cancer. It shows osseous, dural and extracranial components.

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**Fig. 3:** Patient with previous lumbar puncture 2 days ago. (a) Axial enhanced T1WI spin echo shows diffuse pachymeningeal thickening and enhancement within convexities and cerebral falc. (b) Axial 3D T1WI with coronal reconstructions (c and d) showing that enhancement with better definition.

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**Fig. 4:** Normal pachymeningeal enhancement. (a) Axial enhanced T1 spin echo image. Normal dural enhancement is not clearly seen. (b) Axial enhanced 3D T1WI showing pachymeningeal enhancement in both convexities with its features: thin, smooth and discontinuous.

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Fig. 5: Lymphoma. (a and b) Axial and coronal enhanced T1WI spin echo. Extraaxial mass is present with dural, osseous and extracranial components. Leptomeningeal enhancement is seen within the adjacent sulci. (c and d) Axial and coronal reconstructions (T13D sequence with contrast). That leptomeningeal enhancement is better demonstrated.

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Fig. 6: Leptomeningeal carcinomatosis in a patient with breast cancer. Axial and sagittal enhanced T1 spin echo images. Leptomeningeal enhancement is present following cerebellar sulci and the surface of the spinal cord. Metastases is also seen in pituitary gland and infudibullum (b).

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Fig. 7: Postoperative changes. (a) Axial enhanced T1 spin echo image. Patient with recent surgery due to a right frontal abscess. Regional pachymeningeal enhancement is seen. (b) Coronal enhanced T1 spin echo image. Patient two weeks after meningioma surgery. Diffuse pachymeningeal enhancement is demonstrate.
Fig. 8: Patient with history of radiotherapy some years ago. Enhanced T1 spin echo images. Multiple meningiomas. En plaque meningioma within the left convexity (pink arrow).

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**Fig. 9:** Atypical meningioma (WHO grade III). Enhanced T1 spin echo images. A huge frontal mass is seen with parenchymatous and extraaxial-extracranial components and osseous involvement.

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**Fig. 10:** Typical meningioma. (a) Axial T2WI. (b) Axial T1WI spin echo. (c) Axial enhanced T1WI spin echo. (d) Coronal T2WI. (e and f) Coronal enhanced T1WI spin echo. A well-defined mass is present. It shows intense and homogeneous enhancement with dural tail. A small intraosseous component is present.

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**Fig. 11:** Cystic meningioma. Axial and sagittal enhanced T1 spin echo images. Extraaxial mass with cystic component and dural tail.

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Fig. 12: Glyosarcoma. (a) Coronal T2WI. (b and c) Coronal and sagittal enhanced T1 spin echo images. Patient with history of radiotherapy 15 years ago due to Hodgkin’s Lymphoma. Extraaxial mass attached to the dura with dural tail. It shows a deep heterogeneous component that seems to infiltrate the parenchyma.

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Fig. 13: Patient with cutaneous melanoma and cranial metastases. The images demonstrate an extraaxial mass with dural attachment. It is hypointense in T2WI (a) and it shows susceptibility artifact in T2* (b). In T1WI is hyperintense (c) associating a nodule with enhancement within the posterosuperior region (d).

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Fig. 14: Lymphoma. (a) Coronal T2WI. (b and c) Coronal and sagittal enhanced T1 spin echo images. (d) Axial FLAIR-WI. Extraaxial mass with osseous and extracranial components. It shows heterogeneous enhancement and dural tail. FLAIR WI sequence demonstrates meningeal involvement with hyperintensity of the adjacent sulci. This finding is confirmed as a weak leptomeningeal enhancement (b and c).

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**Fig. 15:** Patient with history of fungoid mycosis and secondary CNS involvement. (a) Axial FLAIR WI shows hyperintense areas with mass effect in the left periinsular region and in the left occipital lobe where there is hyperintensity of the sulci. (b) Axial enhanced T1WI demonstrates parenchymal foci of enhancement, leptomeningeal and subependymary enhancement. (c, d e and f). Enhanced 3D T1WI, reconstructions. Leptomeningeal enhancement is better depicted, with nodular appearance in some areas.

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Fig. 16: Cranial metastases of breast cancer. (a) Axial T1 spin echo image shows two blastic lesions in the skull (right frontoparietal region and left parietal region). (b and c) Axial enhanced T1 spin echo image demonstrate irregular pachymeningeal enhancement within the right convexity. (d, e and f) Enhanced 3D T1WI. Dural enhancement is more evident showing in addition the continuity with the affected adjacent bone.

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**Fig. 17:** Dural metastases of breast cancer within the posterior cerebral falx. Enhanced 3D T1WI.

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Fig. 18: Dural metastases in cerebelli tentorium. (a) Coronal T2WI. (b) Coronal reconstruction, enhanced 3D T1WI. Extraaxial mass in the right cerebelli tentorium with homogeneous enhancement and dural tail.

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Fig. 19: Dural metastases of breast cancer with cystic component. (a) Axial FLAIR WI. (b y c) Axial and coronal T2WI. (d) Axial enhanced T1 spin echo image. (e and f) Enhanced 3D T1WI, axial and sagittal reconstruction. Extraaxial mass with solid component that shows dural attachment and dural tail (better depicted in 3D T1 sequences). Cystic components are associated with ring enhancement.

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Fig. 20: Patient with history of resection of glyosarcoma and following radio and chemotherapy. Enhanced 3D T1 sequence, axial reconstruction. Leptomeningeal enhancement with small nodules and masses along the sulci of the right convexity.

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**Fig. 21:** Tuberculous meningitis. (a and b) Axial FLAIR WI shows diffuse hyperintensity of the sulci of both convexities and basal cisterns. (c and d) Axial T1 spin echo image. Diffuse leptomeningeal enhancement with nodular appearance and marked involvement of the basal cisterns.

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Fig. 22: Sturge-Webwr syndrome. (a) Axial contrast enhanced T1 spin echo image showing diffuse leptomeningeal enhancement in the left cerebral hemisfery. (b) Axial contrast enhanced T1 spin echo image demonstrates focal leptomeningeal enhancement of the left occipital lobe, with nodular appearance.

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Fig. 23: Wegener granulomatosis. Axial and coronal enhanced T1 spin echo images show dural thickening and enhancement within the right temporal lobe and cerebelli tentorium.

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Fig. 24: Intracranial hypotension syndrome. (a and b) Axial FLAIR WI shows diffuse dural thickening with small subdural effusions (green arrows). (c and d) Axial T1 spin echo image before and after contrast administration. Pachymeningeal thickening and enhancement in both convexities and cerebral falx.

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Fig. 25: Intracranial hypotension syndrome.

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Fig. 26: Patient with history of cutaneous melanoma in the scalp treated with resection and radiotherapy. 3D T1 reconstructions after contrast administration. Mixed enhancement within the right parietal region with small dural nodules and areas of enhancement within the adjacent sulci. Biopsy confirmed the presence of inflammatory-fibrotic changes, without malignant evidence.

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Fig. 27: Superficial parenchymatous metastases of colorectal cancer. Enhanced 3D T1 images showing a superficial mass with dural contact.

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**Fig. 28:** Patient with type 2 neurofibromatosis. (a and b) Axial enhanced T1 spin echo image. (c) Axial T2WI. Bilateral schwannomas are seen and a small meningioma is present in the left pontocerebellar angle (orange arrow).

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**Fig. 29:** Schwannoma. (a) Axial T2WI. (b) Axial enhanced 3D T1 image. Heterogeneous extraaxial mass in the left pontocerebellar angle with extension to the left temporal fossa and involvement of the cavernous sinus and brainstem.
Table 1: Meningeal Pathology. Diagnostic algorithm. Patterns and questions to be asked.
Table 2: Meningeal Pathology. Diagnostic algorithm. Imaging findings and ethiologies.

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Imaging findings OR Procedure details

A 1.5 Tesla MRI was used to perform the exams.

The MR imaging protocol included systematically all the established sequences for brain examinations completed with sequences after gadolinium administration: axial T1 spin echo and T1-3D acquisition. FLAIR and T1-FLAIR images and fat supression techniques were used in selected cases in which they were necessary.
Conclusion

In the differential diagnosis of meningeal pathology in MR, identification of the pattern of presentation and associated findings, together with the clinical history, can give us the key to establishing the correct diagnosis or determine when biopsy or complementary studies are necessary.