Lumbar spine, climbing the ladder

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Learning objectives

1. Review the approach for the interpretation of lumbar spine MRI
2. Use a common radiological language.
3. Review the causes of low back pain.
Background

The systematic approach of any radiological study allows better learning of the pathophysiology of the disease, giving us more self confidence to address reports and overlook avoiding possible pathologies.

The following sequential evaluation of the lumbar spine, can help this aim with a better understanding of degenerative disease.

1. Alignment
2. Vertebral bodies
3. Discovertebral union
4. Intervertebral disc
5. Facet joint
6. Central canal
7. Retroperitoneum
Imaging findings OR Procedure details

The systematic approach of any radiological study allows better learning of the pathophysiology of the disease, giving us more self confidence to address reports and overlook avoiding possible pathologies.

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1. Alignment
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1.- Alignement

The lumbar spine is an osteofibrocarilaginosa structure that distributes the weight of the body from head to toe. Is a movable non-rigid structure, which also has to accommodate the anteroposterior translation movements, rotary load weights, etc.

Like other elements of nature (Fig 2), the column hast to resist many divergent forces. The best way to do it is adopting a curved structures instead of a linear one, that better match the energy changes. The vertebral bodies are provided, one behind another with uniformly like bricks with cement, but with the difference that between de bricks there are intervertebral disc. Intervertebral discs are like little springs that allows slight movements and and compressed to bear weight.

Despite the continued use in the loading axis of the human body, it determines continuous wear, affecting mainly the weaknesses of the lower back: discovertebral union and facet joints (Fig 3).

The lumbar spine supports the overlying weight level being the last mobile, The lumbosacral joint that supports the greatest. The last lumbosacral space has the greater mobility: 75% of the flexion of the spine. This means that most demanding and generated lumbar diseases at L5-S1 and L4-L5.

To assess the pathology in the alignment can be used both T1 and T2 sequence in sagittal projection.
The physiological lumbar lordosis is between 20° and 60° (Fig. 4), calculated between the top plate of L1 and bottom plate of L5. Smaller angles are considered like lumbar rectification and lordosis greater than 60°, correspond to lumbar hyperlordosis.

Keep in mind that both the lumbar hyperlordosis and rectification can cause back pain (second most common reason that patients seek medical care in the United States) and accelerates the degenerative disease.

LUMBOSACRAL ANGULATION is given by measuring the angle with the horizontal sacrum (Fig 5). When the angle increases, the transaxle increases, causing pain and distension of posterior ligamentous structures overloading facet joint articulation.

The L3 COLUMN LOAD (Fig 6) is obtained by drawing a perpendicular line from the centre of the vertebral body of L3 caudally and measure the distance to the promontory. It is considered pathological distances greater than 1cm. This finding implies a continuous force in anterior lumbar lower elements that favours greater wear on the posterior elements, increasing the chances of listhesis (Fig 7).

Degenerative changes discussed above, help in weakening posterior facet joints, allowing displacement of one vertebral body on another, called listhesis. There are three possibilities the anterolisthesis, the retrolisthesis and laterolisthesi. To find out how to call the displacement is always considered the superior vertebral body motion on the bottom (Fig 8).

To graduate the listhesis, we divide vertebral boy in four, where grad I corresponds to a displacement of less than 25%, grade II is a displacement between 25% and 50%, grade III (50% -75%) and IV (> 75%) (Fig 9).

To assess possible lateral displacements or scoliosis, the MRI locators should be evaluated as coronal sequences are not routinely performed (Fig 10).

2.- Vertebral body

**SIGNAL INTENSITY**: MR imaging appearance of the vertebral marrow depends on the relative proportion of hematopoietic cells and adipocytes within the medullary cavity of the vertebral bodies.

Normal vertebral marrow of the adult shows intermediate signal intensity on T1- and T2-weighted SE images. As a rule, signal intensity of normal lumbar vertebral bodies on T1-weighted SE images must be higher than that of adjacent intervertebral disk in an adult patient (Fig 16).

- Normal variations of vertebral marrow
- Islands of fatty marrow: during adulthood foci of yellow marrow appear in the vertebral bodies which show high signal intensity on T1- and T2-weighted images (Fig 17). CT images of these areas show normal trabecular bone.

- Islands of red marrow: the presence of highly cellular hematopoietic marrow shows areas of low signal intensity on T1-weighted SE images compared to the adjacent marrow. Frequently they are in the periphery of the vertebral body and sometimes central areas of hyperintensity on T1-weighted images are present. Appearance of low-to-intermediate signal intensity on T2-weighted images, absence of bone changes on CT images and at follow-up MR imaging studies help to differentiate this variant from malignant lesions.

- **Focal vertebral lesions that may simulate metastasis**

  - Vertebral haemangioma: is a common vertebral lesion. The signal intensity of vertebral haemangioma on T1-weighted SE images is higher to that of adjacent marrow, but it can also have the same signal intensity and not be visible on T1-weighted images. On T2-weighted images its signal is high. Occasionally, vertebral haemangioma are hypointense on T1-weighted images and can be confused with malignant lesions.

  - Compact bone island: their signal intensity is very low on all sequences. If a high signal intensity rim is surrounding low central signal intensity on STIR images, this lesion is suggestive of sclerotic metastasis.

- **Diffuse hematopoietic marrow hyperplasia**

  Diffuse hematopoietic marrow hyperplasia (Fig 18) or marrow reconversion is observed in several chronic disorders that are associated with anaemia and chronic infections. Red marrow hyperplasia is also seen in heavy smokers. On T1 and T2-weighted images, hematopoietic marrow hyperplasia is heterogeneous because of the presence of residual fatty marrow and areas of red marrow.

- **Metastases (Fig 19)**

  There are two types: lytic and blastic. The most common type is lytic lesions. Frequently they involve the pedicles, appear hypointense on T1-weighted images, lead to cortical destruction and associate soft tissue mass.

  The involvement of other vertebral bodies, the appearance in time and history of malignant tumour are helpful for diagnosis.

  Blastic lesions are markedly hypointense lesions on T1-and T2 weights MR images.

  However, the diagnosis of metastasis is not always so easy, and both, scintigraphy and whole-body diffusion-MR imaging are very useful for the final diagnosis.
The vertebral body is the main part of the vertebra. The anterior part of the vertebral body protects the spinal cord and nerve roots.

To evaluate the vertebral body we essentially consider the vertebral body height, its morphology and signal intensity.

**HEIGHT**: both the vertebral bodies and the discs increase in size from the head to the sacrum. A reduction of the height of a vertebral body of 20% or 4 mm is considered indicative of a vertebral compression fracture.

Lumbar spine injuries are very common in patients with osteoporosis. Common fracture patterns include 'wedge' injuries and 'biconcave' fractures (Fig 11). Biconcave fractures show a collapse of the central area of the vertebral body.

The 'three column model' of Denis (1982) can be used for classification of traumatic lumbar spine injuries. For this author, the stability of the spine is supported by the existence of three columns (Fig 12):

1. Anterior column: Formed by the anterior part of the vertebral bodies, fixed to the anterior longitudinal ligament and anterior portion of the intervertebral anulus fibrosus, and to the upper and lower faces of the adjacent vertebrae.
2. Middle column: Formed by the posterior part of the vertebral body, attached to the posterior longitudinal ligament, and fixed to the posterior part of the anulus fibrosus.
3. Posterior column: Formed by the posterior bony arch: pedicles, laminae, transverse and spinous process, posterior ligament complex, interspinal ligaments, yellow ligaments, and articular capsule.

A fracture is stable and does not require surgical treatment when the lesion is reduced to one of the three columns, generally affects the anterior column. Management of fractures that involve two or more columns depends of neurological symptoms, as these fractures may be symptomatic.

Vertebral bone oedema appears hyperintense on STIR sequences and hypointense on T1-weighted SE images (Fig 13).

**MORPHOLOGY**: the vertebral body has the shape of a cylinder segment flattened front to back, with a concave front and convex rear. Aberrant formation of the spine leads to anomaly of the vertebral segmentation and fusion of the vertebral bodies (Fig 14).
- Segmentation failure: block vertebrae: generally occur in the lumbar spine. Intervertebral disk is absent or rudimentary. Involved vertebrae can be normal in height; often fusion of the posterior elements is seen.

- Failure of chondrification and ossification: butterfly vertebra (Fig 15): failure of fusion of the lateral halves of the vertebral body because of persistent notochondal tissue between them.

3.- Discovetebral union

As mentioned above, one of the weaknesses of the column is the discovetebral union. As wasting progresses vertebral endplates undergo alterations in the bone marrow known as MODIC CHANGES (Fig. 20). These intensity changes, which take wing morphology adjacent to the vertebral endplates, are of 3 types. It is not always easy to distinguish from each other stadiums because there is a trend between them.

Type 1: increased signal on T2 sequence and decreased signal on T1 sequence. If contrast media is used, this Modic change present on uptake of which implies an increase in vascularised tissue, like oedema. This finding may resemble spondylodiscitis, although in these cases the intervertebral disc is hyperintense on T2, there is a marked enhance by the disc and the vertebral body, with poorly defined boundaries between the vertebral body and intervertebral disc. Finally in infections is frequently involved surrounding soft tissue.

Type 2: shows hyperintensity on both T1-and T2. Similar findings to granulation tissue and corresponds to the fatty degeneration of the bone marrow. It is the most common type.

Type 3 is sclerosis of the endplates with hypointensity on T1 and T2.

Another way of discovetebral involvement is INTRAVERTEBRAL HERNIAS or Schmörl nodules (Fig. 21), which correspond to small disc evaginations through the vertebral endplates. These injuries can also associate Modic changes.

What is important about these findings is that by themselves can cause back pain and are more steps on the ladder in the degenerative process overall.

REMEMBER: the top differential diagnosis of Modic changes is spondylodiscitis because it is similar to Type 1 changes but not equal. In spondylodiscitis there hyperintensity on T2 intervertebral disc, importante oedematous changes of the adjacent vertebral bodies, that are larger than in Modic changes. Finally, usually there is a collection on soft tissue or oedema of surrounding soft tissue (Fig 22).
4.- Intervertebral disk

One of the most common causes of back pain in the population, are degenerative changes in the intervertebral disks, lumbar discogenic pain, which is a low back pain without radiation. The disc annulus is innervated by recurrent meningeal nerves and small ventral branches of somatic nerves.

MRI is indicated to assess the technical disc pathology. The sequence that best values the pathology is the sagittal T2. The normal disc has a centre hyperintensity, which corresponds to the nucleus pulposus, and a periphery which is hypointense annulus. As the disease progresses the intervertebral discs become dehydrated, losing the elastic properties, and lose the signal in T2 (Fig 23) to be completely hypointense.

In the degenerative process as disc dehydration progresses, the discs lose their height. Pearce et al classified disc degeneration on sagittal T2. This classification can be tedious for routine clinical practice, but it is a clear picture of the evolution of disc disease. All these findings are described globally as DISCOPATHY.

<table>
<thead>
<tr>
<th>GRADE</th>
<th>DIFFERENTIATION OF NUCLEUS PULPOSUS FROM ANULUS</th>
<th>SIGNAL INTENSITY OF NUCLEUS PULPOSUS</th>
<th>DISK HEIGHT</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>YES</td>
<td>HOMOGENEOUSLY NORMAL HYPERINTENSE</td>
<td>NORMAL</td>
</tr>
<tr>
<td>II</td>
<td>YES</td>
<td>HIPERINTENSE WITH HORIZONTAL DARK BAND</td>
<td>NORMAL</td>
</tr>
<tr>
<td>III</td>
<td>BLURRED</td>
<td>SLIGHTLY DECREASED, MINOR IRREGULARITIES</td>
<td>SLIGHTLY DECREASED</td>
</tr>
<tr>
<td>IV</td>
<td>LOST</td>
<td>MODERATELY DECREASEAED, HYPOINTENSE ZONES</td>
<td>MODERATELY DECREASED</td>
</tr>
<tr>
<td>V</td>
<td>LOST</td>
<td>HYPOINTENSE</td>
<td>COLLAPSED</td>
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The most interesting thing about the board ahead is that disk degeneration begins much earlier than is commonly believed (complete hypointense nucleus pulposus).
ANNULAR TEARS are described as T2 hyperintensity (Fig 24) in the posterior disk region or disc focal uptake of contrast. Annular tears represent fluid or mucoid material from the nucleus pulposus.

Annular tears indicate a reduction in the stiffness of intervertebral disk in increased motion of that lumbar segment. This instability transfers new rotational and axial forces to connective tissues and vertical pedicles.

Other degenerative effects on the intervertebral disc are "exvacuo" phenomena consisting in forming gas (nitrogen) as degenerative waste product. In RM can be observed as hypointense on T1 (Fig 25), but they are better seen on CT.

Finally hyperintense T1 foci on intervertebral disks can be also seen. Most of the time they correspond with dystrophic calcifications, although they are better depicted on CT or even in plain radiograph.

In the progression of the degenerative disease, intervertebral disks lose their smooth peripheral surface and develop lumps. From radiological terminology should disappear the word "hernia" because this word means herniation of the nucleus pulposus and can make more difficult patient-surgeon relationship with misleading terms.

Each hospital should agree with spine specialists, orthopaedic surgeons or neurosurgeons, to choose the same terms to try to speak all the same language and avoid misunderstandings. We recommend using the recommendations of the North American Society of Spine, Spine Radiology and Neuroradiology 2001.

• **Bulging disc:** Disc general increase beyond the vertebral border.
• **Protrusion:** the greatest distance, in any plane, between the edges of the disc material beyond the disc space is less than the distance between the edges of the base, in the same plane. The base is defined as the cross-sectional area of disc material at the outer margin of the disc space of origin. In the cranio-caudal direction, the length of the base cannot exceed, by definition, the height of the intervertebral space. It can be divided into focal if it affects less than 50% of the circumference or diffuse if more than 50% in axial plane.
• **Extrusion:** in at least one plane, any one distance between the edges of the disk material beyond the disk space is greater than the distance between the edges of the base, or when no continuity exists between the disc material beyond the disc space and that within the disc. When in sagittal plane the disk exceed intravertebreal height (Fig. 26).
• **Migration:** intervertebral disk displacement in space pretecal cranial or caudal fat remaining in contact with the original disk.
• **Sequestration:** when the displaced disc loses the link with the original intervertebral disc.
5.- Facet joint

The facet joints (FJ) are paired synovial joints at the posterior aspect of the spinal column. Each joint consists of the articulation between adjacent superior and inferior articular vertebrae process (Fig 28).

FJ guide spinal flexion-extension movements and prevent excessive rotation.

Due to their rich innervations, FJ have an important role in low back pain, and are often underestimated in everyday MRI inform.

FJ degenerative changes include osteophyte, articular processes hypertrophy, subchondral osteosclerosis with cyst formation, articular space thinning, "ex vacuo phenomenon", joint effusion, ligamentum flavum and interspinous ligamentum hypertrophy or calcification. CT is the best imaging for detecting bone changes such as osteophyte, sclerosis or subchondral cyst formation, but MRI can help in soft tissue changes.

   - The key findings on MRI are:

- FJ articular space thinning: less than 2 mm.

- Ligamentum flavum hypertrophy: on axial plane > 4mm. On sagittal plane can also be seen as posterosuperior foramina obliteration (Fig 29). Intra ligamentum flavum cysts have also been described.

- FJ effusion: be aware of axial T1, that also de fat is bright on T2 (Fig 30)

- FJ synovial cyst are hyperintense on T2 and equal or slightly greater on T1. Some cysts have haemorrhagic content and therefore they can be hypointense on T2. Thin peripheral contrast enhancement also can be seen. The next step about FJ synovial cyst are they location. Some of them are located anteromedial to FJ and hence they can help to foramina or central canal stenosis (Fig 31). Other cysts are located on paravertebral tissues and could be infected.

- Facet subluxation: FJ degenerative disease and aging can help to damage the joint capsule increasing ligamentous laxity, that guide to a facet subluxation, with anterior and lateral slip of the inferior articular process that can cause foraminal stenosis and help to spondylolisthesis.
- Pedicle fat infiltration: high T1 signal on inferior pedicle border is one of the first changes on degenerative disease.

- Interspinosinal process effusion, interspinosum flavum hypertrophy,

Finally, **Baastrup disease** (Fig 32) is clumping spinous processes, with osteophyte formation, interspinous ligament hypertrophy and even bursitis that allow neoartrosis between spinous processes; reaching them in touch with neoartrosis forming between spinous processes. These changes are also source of LBP. Unfortunately, this is another cause of low back pain.

FJ degenerative disease is best seen with sagital and axial STIR sequence, because pedicle or spinous process oedema, FJ effusion or synovial cyst on paravertebral tissue are better depicted.

6.- Central canal

**Canal stenosis** can be of three types: **congenital, acquired or mixed**.

There are three location of stenosis: **central canal, lateral recesses and foramina** (Fig 33).

Clinically canal stenosis corresponds with radicular pain in lower limb and neurogenic claudication (pain when walking that subsides with the sitting position). The important thing about this disease is that it is the main indication for surgery in adults older than 65 years.

The anteroposterior diameter of the bone margins measured parallel to the disc is superior to 15mm, interpeduncular distance must be greater than 18mm and the channel AP distance must be greater than 4-5mm (Fig 34).

The **central canal** stenosis is characterized by circumferential decrease "in napkin ring" in the sagittal projection, with a smaller area of 1.5 cm2 or anteroposterior diameter less than 11.5 mm.

In clinical practice the assessment of stenosis is done by visual measuring, however it should be noted that these measures can be helpful for those cases where we have doubts.

The fundamental cause of stenosis in the adult population is the degenerative process, mainly derived from the productive changes in the posterior elements.

**Lateral canal** can be divided into three (Fig. 33):
Zone 1 or lateral recess.
Zone 2 or foraminal.
Zone 3 or extraforaminal.

It is very important is to assess foraminal stenosis as it is a common cause of low back pain, both the irradiated and non-irradiated. The best assessment of this segment is performed in sagittal, being useful both T1 sequence where periradicular fat can be measured as the T2 (Fig. 35).

**Cord, conus medullaris and cauda equina nerve roots:** proper assessment of these structures serves to rule masses (intramedullary or extramedullary). We should get used to assess the level of the conus medullaris, as well we routinely discard the tethered cord syndrome (Fig 36) when it is below L1-L2 or less thickening of the filum terminale 2mm.

We must also assess the distribution of the nerve roots; They flow individually, with posteroanterior direction in sagittal projection. Grouping or adhesions of them may indicate the possibility of arachnoiditis.

Radiculitis is inflammation of a nerve root that can be caused by multiple aetiologies, the most frequent degenerative. The most common findings are an enlarged and hyperintensity on T2 (Fig 37). Other findings that can be detected are increased signal on STIR or root contrast enhancement.

We must take into account variations in the configuration of the cauda equina, the most common joint output nerve roots. This anomalous course may condition or not, the unexpected appearance of symptoms.

**7.- Retroperitonum**

Last but not least important in the lumbar MRI is to study visible retroperitoneum. Mon radiologists do not usually pay much interest to this location because they go straight to what interests them: stenosis, "hernia", etc.

Unfortunately for radiologist it is often also an empty area due to overwork or lack of practice in pathology at that location area. A good reading systematic report will enable us not to overlook and gives a touch of quality to our report.

The main areas visualized are kidney, retroperitoneum (aorta, inferior vena cava), bladder-prostate in men and ovaries-uterus in women (Fig 38-40).
Fig. 2: Distribution of force loads due to arcs. Architectural Elements to exemplify how the column due to its curved structure helps to dissipate energy.

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**Fig. 3:** Loads of force on the weak elements of the column: discovertebral union and posteiores elements (facet joints)

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Fig. 4: Lumbar lordosis angle: from upper plate of L1 to lower plate of L5.
Fig. 5: Lumbosacral angle: The anterior ramp slope of sacral bone (S1). It involves a continuous force, that the posterior elements and paraspinal muscles have to counteract.

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Fig. 6: Method of measuring the L3 column load.
**Fig. 7:** Lumbar hyperlordosis: Increased lumbar lordosis, lumbosacral angle and anterior displacement load column. The load forces on the column above are superior to the rear ones, which will condition with time, wear and subsequent anterolisthesis.
Fig. 8: The previous weight bearing has overcome the rear pillars. It produces anterior displacement of L4 on L5. ANTEROLISTHESIS.

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Fig. 9: Division of listesis versus displacement of the vertebral bodies. Each grade is 25%
Fig. 10: MRI Locator, which shows a scoliotic curve to the left. The locator can help to guide us in the sagittal and axial, as many times in scoliotic curves is complicated numbering the vertebral bodies.

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Fig. 11: Fracture types: a) Normal vertebral body b) Fracture-wedging c) Fracture biconcave d) Fracture-crushing

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Fig. 12: Division of spines: anterior column- medium column - posterior column

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Fig. 13: Vertebral body fracture in lower plate with associated vertebral edema. The image on the left corresponds to STIR sequence that shows generalized hyperintensity on vertebral body, which corresponds to hypointense T1 sequence.

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Fig. 14: Normal column on sagittal T1. Superimposed in yellow boxes, embryonic sclerotome segments, and red lines correspond to intersegmental arteries. During the further development escleromas are divided into two, joining with the corresponding cranial or caudal half, leaving the artery in the back half of the vertebral body. The separation line will become sclerotome intervertebral disc

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**Fig. 15:** Coronal T2 sequence, showing right convex scoliosis (pink line), secondary to hemivertebra (yellow circle).

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**Fig. 16:** T1 hyperintense foci in individual vertebral bodies correspond to fat. The most common finding of hyperintensity on T1

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Fig. 17: Hyperintensity on T1 and T2, from L5 and sacrum, without changes in STIR. Fatty replacement secondary to radiation.

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Fig. 18: Generalized hypointense on T1 vertebral bodies, secondary to anemia.

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**Fig. 19:** Multiple T1 hypointense lesions affecting spine. This findings should make us suspect the possibility of metastasis. Pedicle involvement (arrows) strengthens us our diagnosis.
Fig. 20: Modic Changes

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**Fig. 21:** Intravertebral herniation, the images can be seen as part of the disc bulges in the lower plate. The findings and is telling us that there is degenerative disease.

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**Fig. 22:** Spondylodiscitis. Marked hypointensity on T1-L5 and sacrum. To differentiate Modic changes: 1. - Hyperintense disk (green arrow) 2. - Marked vertebral edema, too extensive to Modic changes (red arrow) 3. - Marked contrast enhancement, including soft tissue (purple ring).

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Fig. 23: Discopathy evolution (red ring): 1 normal disk, 2 the disk gradually loses T2 signal and then begins to lose height 3, and 4 in later phases is lost almost the entire disc.
Fig. 24: Posterior annular rupture. Remember that initially shines on T2, CSF-like but the hyperintensity gradually is lost.

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**Fig. 25:** Exvacuo Phenomena: hypointense linear images within the discs on T1.

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Fig. 26: T2 sagittal image, eg where the extrusion material is subsequently protrudes more than the height of the disc.
Fig. 28: Posterior elements: -white line: joint. -red lines: articular facets. -green line: yellow ligaments. -green-dashed line: spinous process.

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**Fig. 29:** Sagittal T2: on the left, green arrow points to the normal yellow ligaments, while on the right arrow shows the typical pathological bulge when you start with ligament hypertrophy.

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**Fig. 30:** Example of facet synovitis that is displayed in both sagittal and axial.

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**Fig. 31:** Example of right facet synovial cyst. Keep in mind that the synovial cyst can be placed anywhere round the joint region, but when it is located medial to the facet joint it can determine lateral recess stenosis, as in this case.
**Fig. 32:** Bastrup Disease: -purple arrow, spinous "kissing" -white asterisk, hypertrophy interspinous ligaments -interspinous liquid, green arrow.
Fig. 33: Blue: lateral recess Yellow: foraminal region White: region extraforaminal

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**Fig. 34:** Distances:
- Red: > 4-5mm
- Blue: > 15mm
- Green: > 18mm

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**Fig. 35:** Foraminal stenosis: the image on the left corresponds to the normal morphology foraminal fat. As the disease progresses degenerative (disc and posterior elements correspond to the arrows) decreases fat as indicated by the red arrows. The fat decreases until cleared periradicular fat. Severe stenosis. The assessment should be done in stenoses T1 for overrating them.

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Fig. 36: Tethered cord: Cone L2 spinal below (red line) anchored to a lipoma (blue diamond).

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Fig. 37: Enlargement of left root wiht entrapment due to degenerative changes in the posterior elements. RADICULITIS

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**Fig. 38:** Asterisk: example of adrenal adenoma. SEARCH RENAL FOSA

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Fig. 39: Example of a horseshoe kidney. SEARCH RETROPERITONEUM.

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Fig. 40: In the image on the left, you can see fibroids and diverticula. In the image at right, in white frame shows a strong involvement lumbar spondylodiscitis. Unfortunately the patient had two diseases, see the red arrow corresponded to a polyp and hematometra that turned out to be an adenocarcinoma. REVISE THE PELVIS.

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Conclusion

1. The approach to lumbar spine MRI has been provided using a "step by step" schedule.
2. Common radiological nomenclature has been suggested.
3. A review on low back pain has been done, focusing on degenerative facet joint disease and biomechanical forces.
References


Remy S. Nizard, Marc Wybier, and Jean-Denis Laredo. Radiologic assessment of lumbar intervertebral instability and degenerative spondylolisthesis. RADIOLOGIC CLINICS OF NORTH AMERICA VOLUME 39. NUMBER 1 JANUARY 2001. 55-71


