10 DEGENERATIVE DISK DISEASE IN LUMBAR SPINAL COLUMN

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The intervertebral disc, which contributes the stabilization and flexibility of spinal column (……..) (omurga kolononun), has complicated structure formed by ligament. The intervertebral disc consists of three main structures that are "cartilage end-plate", "nucleus pulposus", and "annulus fibrosus". The cartilage end-plate is composed of the fibrocartilaginous peripheral region that frames apophyseal rim, and the centrally-located hyaline cartilage that covers the surface of intervertebral disc. The border between central and peripheral regions is mostly ambiguous [1, 2]. There are any numbers of pores that have vascular structures in vertebral end-plates, and these pores play a crucial role in the nutrition of intervertbral disc.

Nucleus pulposus converts the power formed as a result of axial pressurization into radial power, and it enables the distribution of this radial power to end-plates homogenously. Nucleus pulposus consists of type-II collagen that has more irregular alignment than annulus fibrosus, and matrix that includes hyaluronic acid and glucosamine. Type-II collagen is more stable against compression than type-I collagen. Nucleus polposus, which has semi-liquid structure in infants and young adults, dehydrates by aging. The border between nucleus polposus and collagen fibers of annulus is considerably apparent in newborns. This border becomes ambiguous in the disc that had converted into its adult form after puberty. In adults, the disc is monitored as intranuclear cleft that has transverse orientation at its central region, and that composed of dense collagenous and elastic fibers (Yetişkinlerde disk, santral kesiminde transvers oryantasyon gösteren ve yoğun kollajenöz ve elastik liflerden oluşan intranükleer kleft izlenmektedir) (Figure 1).

Figure 1: Sagittal section, T2 weighted image. In young patient, the discs, which have normal signal density and configuration, are seen. The hypointense linear signal pursued at central region of disc shows consistency with nuclear cleft.

Annulus fibrosus, which forms the most of the intervertabral disc, performs as a restrictive barrier for nucleus polposus. "Annulus" consists of two type fibers that are fibrocartilaginious having ring-like and concentric arrangement, and collagenious. Inner plate composed of fibrocartilaginious and collagenious fibers cleaves into end-plate.

Outer layer formed by type-I collagen fibers clings to vertebral rim and periosteum. Peripherally-located collagenious fibers also get involved in anterior and posterior ligament. Annulus fibrosus is stronger at the front side rather than at the back side. As a result of studies on cadaver, it is revealed that there is dense vascularity at the region where peripheral annulus cleaves into vertebra [3]. In children and young adults, the disc structure is nourished by deep perforating vascular structures. However, it is seen the rarefaction of vascularity, and also the decrease of its penetration, by aging.

In adults, vascularization of nuclear pulposus is limited with only nutritive veins that reach the outer annulus fibrosus. The nutrition of other regions of disc is made by means of diffusion of metabolites. As a result of this restricted nutrition, discs can be easily damaged metabolically or mechanically, and exact healing of discs mostly can not be possible. Disc damage can cause the decrease of intradiscal pressure resulting with the decrease of disc level, the formation of annular tear, and the herniation of disc.

1. Degenerative Disc and Degenerative Disc Disease

The concept of "degenerative disc", which is accepted as physiologic, describes the alteretions that emerge by aging. On the other hand, "degenerative disc disease" is clinical picture that arises as a result of expedited degenerative changes, and causes symptoms at early ages.

It is very hard to differentiate between structural changes in intervertebral discs by aging and degenerative disc disease, even it is mostly impossible. There two degenerative processes related with intervertebral disc, which are spondylosis deformans, and intervertebral osteochondrosis. The first of them mostly affects the annulus fibrosus and adjacent apophysis, whereas the second one affects nucleus pulposus and end-plates. In intervertebral osteochondrosis, there can also be watched the widespread annular tear resulting with atrophy [4]. It is accepted that spondylosis deformans emerge as a result of normal aging; however intervertebral osteochondrosis is not always symptomatic but pathologic.

Spondylosis deformans: With increasing age, the amount of mucoprotein ("proteoglycan") decreases, and the ratio of ceratin sulphate to chondroitin sulphate increases. Another important variation that emerges by aging is increasing collagen that causes the disc to have fibrotic structure. All of these changes make way to decrease in the water-holding capacity of intervertebral disc, and in hydrostatic pressure.

It is considered the loss of signal intensity in T2-weighted images in the range of low to moderate during central disc localization as a change related with aging; and there are similar images for all discs in the examination region (Santral disk lokalizasyonun T2 ağırlıklı serilerde hafif-orta derecede sinyal kaybı yaşlanmayla ilişkili değişiklik olarak değerlendirilir). However, it is preserved the intervertebral disc level and the regular borders of disc. It can be observed the protrusion of symmetric disc as an outcome of the reshaping of osteoporotic vertebras by aging [7] (Figure 2a).

The formations of anterior or lateral osteophyte present in almost all over 40-year old population, and this situation is accepted as normal aging symptom. Osteophytes locate 2-3 mm away from the discovertebral joint, where Sharpey fibers cleave; and they have horizontal orientation.
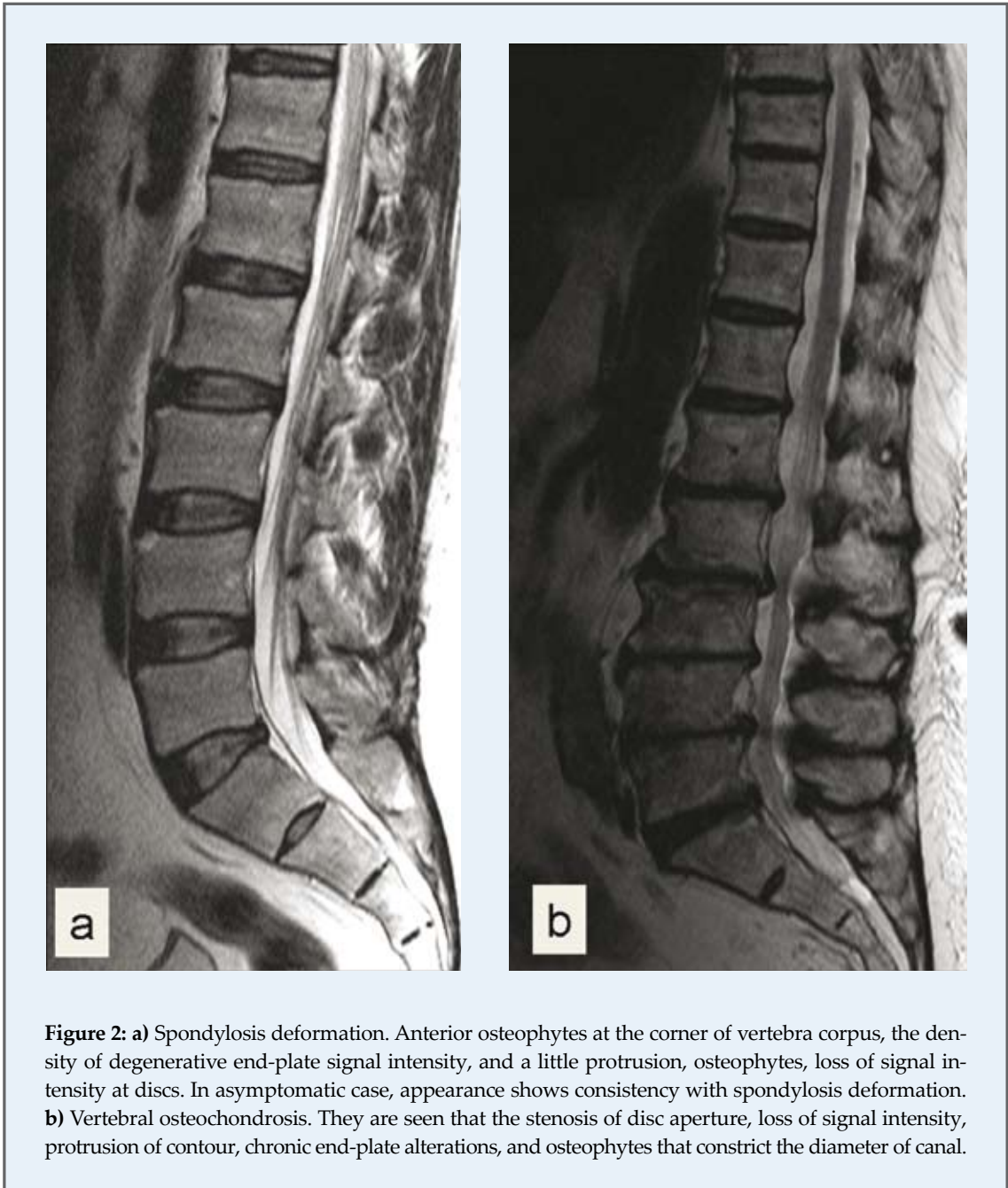
Beginning from third decade, little concentric or transverse fissures can be developed [8, 9].

Vertebral Osteochondrosis: Radiographically, it is characterized with the stenosis of intervertebral osteochondrosis disc aperture, the irregular contour of protrusive disc, the osteophytes that elongate to central spinal cord and foramens, the sclerotic end-plates, and the changes of chronic vertebra corpus marrow.

However, the disc level is variable at lumbosacral joint depending on segmentation differences, and the decrease of disc level does not signify osteochondrosis in every case. The magnetic resonant (MR) imagining is enough sensitive to detect the early symptoms of intervertebral osteochondrosis.

In intervertebral osteochondrosis, normal disc tissue is entirely replaced by fibrosis; and, the loss of signal intensity in T2-weighted images and the disappearing of the line that specifies the margent of annulus fibrosus emerges. In further degeneration, the decrease of disc level, the disorder and protrusion in disc contour is detected.

The reactive marrow changes at cartilage end-plates often accompany with further degenerative disc variations (Figure 2b). The radial annular tear was detected in too few of over 40-year old cases by autopsy studies; therefore, this symptom is accepted as a symptom of "vertebral osteochondrosis" [9]. The gas, which is seen during central disc localization, is almost always a symptom of intervertebral osteochondrosis [10].

Figure 2: a) Spondylosis deformation. Anterior osteophytes at the corner of vertebra corpus, the density of degenerative end-plate signal intensity, and a little protrusion, osteophytes, loss of signal intensity at discs. In asymptomatic case, appearance shows consistency with spondylosis deformation. **b)** Vertebral osteochondrosis. They are seen that the stenosis of disc aperture, loss of signal intensity, protrusion of contour, chronic end-plate alterations, and osteophytes that constrict the diameter of canal.

2. Degenerative Disc Variations in Anterior Elements

2.a. Degenerative End-Plate Variations

In degenerative disc disease, the signal variation at adjacent vertebras' end-plates is frequent symptom. By radiography and computerized tomography (CT) imagining, the increase of density named "discogenic sclerosis" is seen at vertebra end-plates which are affected during degenerative process.

In MR imagining, the signal alteration of degenerative vertebra end-plate is collected under three main groups by Modic, and its clinical significance is questioned for more than two decades [11]. It is thought that type 1-variation of end-plate stems from inflammatory, and the end-plate exhibits the hypointense involvement in T1-weighted images, the hyperintense involvement in T2-weighted images, and the involvement of contrast in postcontrast images (Figure 3). In histopathological study, hypervascularity are seen at subchondal bone, and fissures are pursued at cartilage end-plate. Type-1 signal model is related with the acute degenerative disease, segmental instability, and disc hernia [12].

There is a strong correlation between type-I degenerative alteration and back ache, it is stated that the fusion operation can be helpful to patients who have type-I alterations [13]. A little part of Type-1 Modic alteration disappears whereas the big part of it proceeds to type-II Modic alteration.

In chronic degenerative diseases, type-II alteration, which resembles paravertebral lipid tissue, and which is thought that to be characterized with the lipoid marrow replacement at stabile spinal column, exhibits the hyperintense signal model in T1 and T2-weighted images (Figure 4). (Kronoik dejeneratif hastalıklarda ve stabil spinal kolonda yağlı kemik iliği replasmanı ile karakterize olduğu düşünülen ve paravertebral yağ dokusuna benzeyen T1 ve T2 ağırlıklı serilerde hiperintens sinyal örneği sergileyen Tip II son plak değişikliği izlenmektedir.)

It is stated that type-II alteration can be an outcome of chronic ischemia [14]. However, some studies suggest that Modic type-II signal model, which is known as silent, has equal capacity to form a symptom with Modic type-1 alterations [12]. In chronic degenerative disc disease, as a response to the changes in intercurrent stress, it is possible to return from type-II signal model to type-I signal model. Type-III end-plate signal alteration, on the other hand, is characterized with sclerous, and its pathogenesis is not completely understood.

Sclerotic end-plate exhibits hypointense signal in T1 and T2-weighted images (Figure 5). By the studies on asymptomatic volunteers, degenerative end-plate signal alteration is detected in the
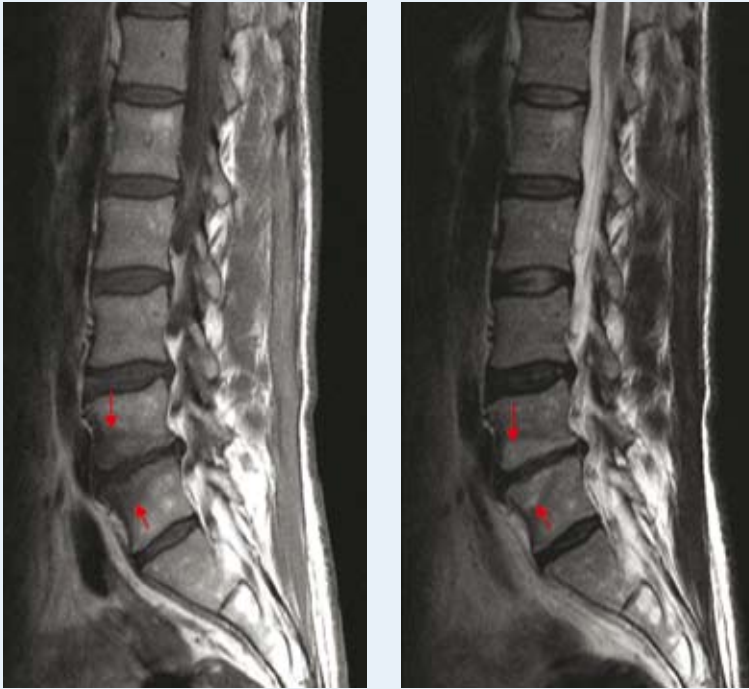

Figure 3: Modic Type I end-plate signal alteration. They are seen that T1 hypointense at the reciprocal end-plates, T2 hyperintense signal intensity, and in the compliance with degeneration, the loss of signal and level at intervertebral disc within range of L4-L5.

range of 10-15%. Degenerative end-plate signal alterations, which do not cause a symptom, generally locate at the upper lumber region and at the anterosuperior end-plate as focal. In asymptomatic cases, significant degeneration is not be detected at adjacent intervertebral disc, unlike symptomatic cases [15].

2.b. Annular Tear

Although the main area of interest of radiologic operations is lumbar roots, ingredients of thecal sac, and the relationship between disc and neural structures for patients who consult the doctor with complaints of back ache; neural element compression is not detected in most of the patients. By the provocative discographic investigations that are made due to annular tear,
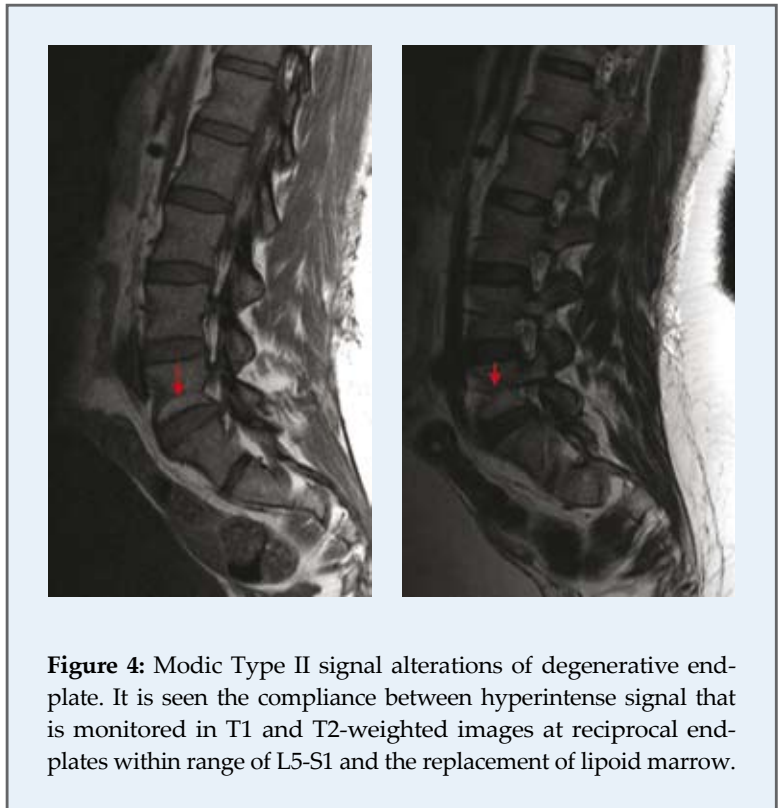

Figure 4: Modic Type II signal alterations of degenerative end-plate. It is seen the compliance between hyperintense signal that is monitored in T1 and T2-weighted images at reciprocal end-plates within range of L5-S1 and the replacement of lipoid marrow.
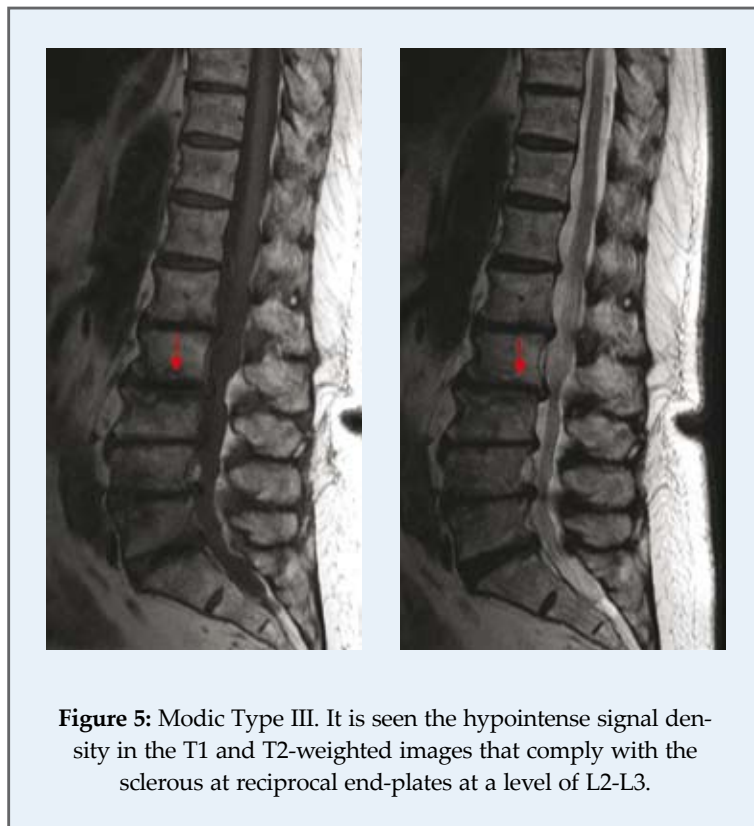
although there is no root compression, they can be often seen the pain that spreads to gluteal area, sciatic, inguen, and proximal upper extremity [16]. In degenerated intervertebral discs, the peripheral high-intensity region, which defines T2 hyperintense focuses where annulus fibrosus locates, was firstly described by Aprill and Bogduk in 1992 [17]. By the first studies related with the peripheral high-intense region, it is stated that this symptom corresponds enflame annular tear within the range of third and fifth that had detected in discography; and this symptom is sufficient to determine the disc degeneration that causes to pain [18]. Although there are some studies that suggest that there is close relationship between the peripheral high-intensity region and the formation of pain in discography [19, 20], its clinical significance is

Figure 5: Modic Type III. It is seen the hypointense signal density in the T1 and T2-weighted images that comply with the sclerous at reciprocal end-plates at a level of L2-L3.

questionable because it is detected the annular tear by the high rate reaching 38% in the group of asymptomatic patients, and because the provocative discography always detects the annular tear in highly degenerative discs.

It is accepted that the peripheral high-intense region usually emerges with the degenerative alterations of nucleus polposus, and that region arises from biomechanical impairment. The granulation tissue that comply with the annular tear exhibits hyperintense signal at the near of cerebrospinal fluid (CSF) in the thecal sac in the T2-weighted images, and the patient who is injected the contrast agent holds this contrast agent in (postcontrast) images. This signal density and involvement of contrast agent are owned by the vascularized granulation tissue in the peripheral annular tear. The most sensitive sequence in deciding of the peripheral high-intense region

Figure 6: At sagittal plane **a)** T2 weighted **b)** Post-contrast T1-weighted fat-suppressed series. Punctate T2 hyperintense signal and peripheral contrast enhancement is compatible with high-intensity zone at L4-L5 intervertebral distance. At localization of the conus medullaris space-occupying lesion consistent with cavernoma.

is T1-weighted images of patients who had injected contrast agent (Figure 6). The density of peripheral high-intense can endure for long years; and it does not always correspond to acute or subacute tear. It is regarded necessary to make discography before surgical intervention because the 13-14% of peripheral high-intense region is not related with clinical complaints. (Periferal yüksek intensite bölgesi, % 13-14'ü klinik yakınmalar ile ilgili olmadığı için cerrahi girişim öncesi diskografi yapılması gerekli görülmektedir.) Annular tears are classified into three main groups that are radial, vertical ("concentric"), and peripheral border tears. Radial tears develop as a consequence of the elongation of fractures that emerge at nuclear cleft to peripheral annular fibers in the orientation of radial or oblique. Generally, they locate at the posterior or posterolateral region of L4-L5 and L5-S1 intervertebral discs (Figure 7a).

Radial tears sometimes join to pre-existing peripheral border or vertical tears, and they constitute the peripheral high-intense region. The importance of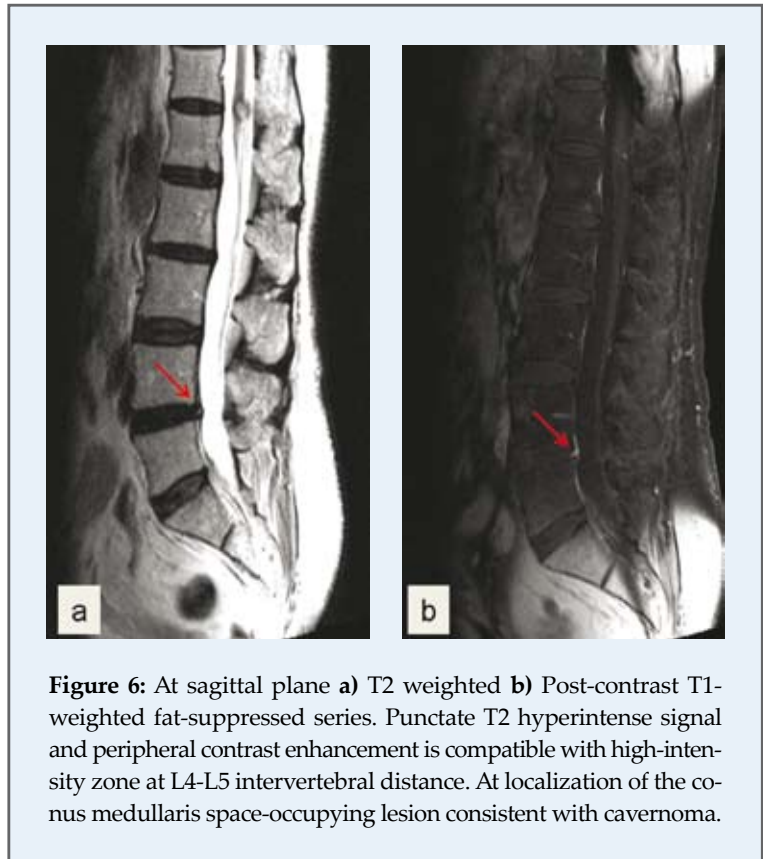 radial tears is that they are precursor lesion of disc herniation and they causes back ache. However, all radial tears do not cause back ache. One of the theories, that are accentuated to explain the endangering of radial tears to back ache, is that these tears affect the ache-sensitive nerve fibers which locate in the annulus layers of nucleus sliding to outer annulur layer by chemical irritation and inflammation caused by them. Another theory suggests that hence degenerative nucleus can not distributes the axial pressure to outer annular fibers, outer annular fibers are obligated to resist the redundant pressure, and this situation irritates the inflame nerves that locates there. When radial tears are analyzed by microscope, there occurs granulation tissue that exhibit vascularization. This granulation tissue performs a duty as barrier against the peripheral elongation of tear, and the sliding of nucleus. Since nucleus has no direct vascularization, the granulation tissue almost always locates at the 1/3 outer annulus, and precise recuperation does not occur [22]. The partial recuperation of radial tears explains the recurring of back ache occasionally. The vertical tears are the most frequent type of annular tears, and

they are detected in advanced ages and juveniles similar frequently. The vertical tears, which generally locate at 1/3 outer and medial annulus fibrosus, arise from the delamination caused by the accumulation of mucoid agent or liquid through the annulus layer (Figure 7b).

Trauma plays a role in etiology, and the overload with torsion is triggering factor. Hence outer annulus has rich sensorial nerve nets, these tears can cause pain. Peripheral border tears, on the other hand, generally locating at anterior are horizontal tears, and they emerge at the place where outer annular layer cleaves periosteum (Figure 7c). Generally, little osteophytes accompany with peripheral border tears. By the studies on animals, it is shown that the peripheral border tears stimulate the degenerative disc disease, end-plate degeneration, and even facet arthrosis [23, 24]. This type of degenerative incidents is closely related with pain.

Intervertebral disc degeneration can cause the pain impairing the quality of life, and a lot of patients can get rid of this pain by the fusion, replacement of total lumbar disc, and dynamic or fusionless stabilization operations that are came into use prevalently in recent years. Therefore, it is important to determine the level of symptomatic intervertebral disc before surgery. While the most reliable diagnostic procedure is provocative discography, there are some publications that state that this method has false-positive rate within the range of 20-40% as well [25, 26].

The success of operation varies within the range of 40-60% in the group of discogenic patients who had been examined by discography before surgery [27]. The accepted system in classification of radial tears is Dallas discogram classification [28].

After a while later the injection to central region of suspicious intervertebral disc, the patient is placed into the computed-tomography, and the axial images are obtained. The contrast agent fills the tears that form from the central region of disc through annulus. These tears, which involve nucleus
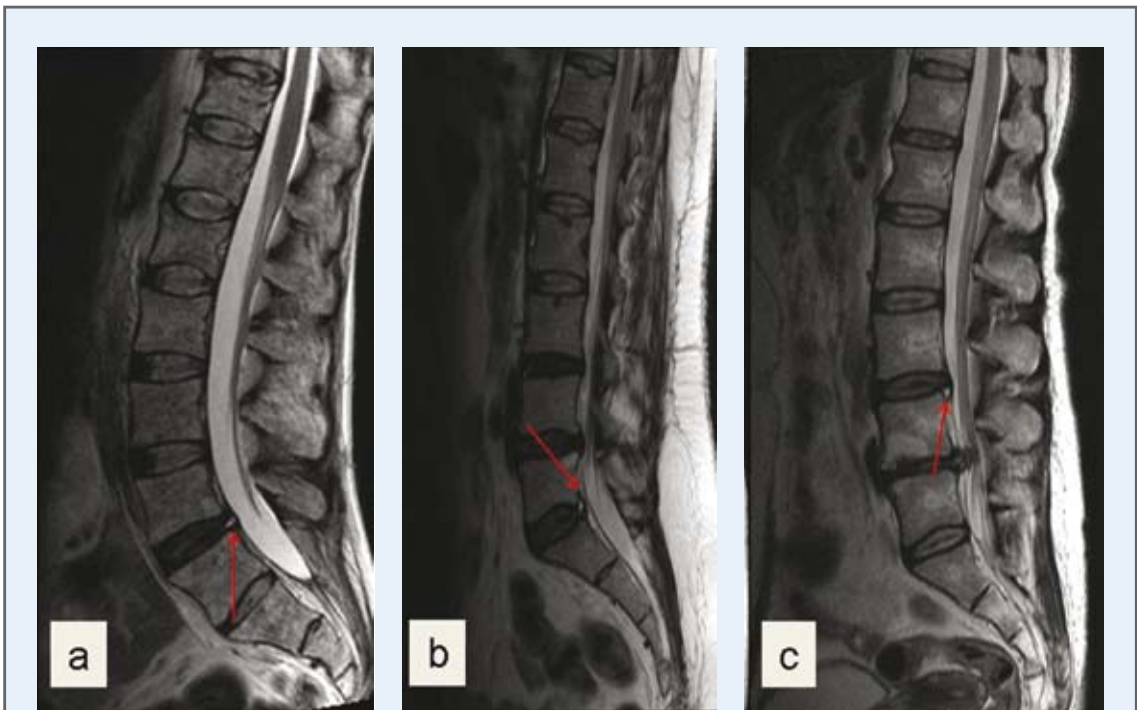

Figure 7: a) L5-S1 annular radial tears extending from the inner annular fibers to the periphery. **b)** annular vertical tear of outer annular layer at L5-S1 distance. **c)** Peripheral annular tear where outer annular layer attached to the periostum at L3-L4 distance, and at L4-L5 distance there is extrude transligamentous herniation.

pulposus and annulus, are classified in six groups with respect to their degree. The zeroth degree corresponds to normal disc. In the first degree tears, the contrast agent spreads to 1/3 inner annulus fibrosus; however, it spreads to 1/3 mid-annulus fibrosus in the second degree tears. In the third degree tears, the contrast agent goes ahead peripheral layer by passing through inner and mid- annular layers. In the fourth degree tears, the contrast agent reaching the peripheral layer combines with the vertical tear that emerges with the radial tear, and then it spreads around of the disc at least 30°. In the fifth degree tears, the integrity of Sharpey fiber is ruined as well, and the contrast agent spreads to the out of the disc (Figure 8). This type of tear causes the chemical radiculopathy.

2.c. Disc Herniations

One of the steps required for providing the most proper treatment to the patient who consults the doctor with the complaint of back ache, is that radiologists and clinicians committing the treatment must compromise on the same nomenclature and classification system. A lot of interchangeable nomenclature and classification cause confusion at the present time. The standard description was published by North American Spine Society/NASS in 2001.

The intervertebral disc herniation implies the translocation of nucleus pulposus, end-plate cartilage, fragmented aphophysal bone, or annular tissue to the out of the normal disc border. This replacement is local, and hernia disc involves the region smaller than 50% of all disc region. The notion of "Annular apophysis", on the other hand, is used for the translocations related with the more than half or the entire disc region, and it does not exceed 3 mm. Herniation is subclassified as protrude and extrude disc with respect to the configuration or amount of disc. The protrude discs do not elongate to the out of the planes of superior or inferior end-plate at sagittal plane. If protrude disc is related with the smaller area of 25% of entire disc region, it is named "focal"; if it is related with the 25-50% of the entire disc region, then it is called "broad-based" (Figure 9).

On the other hand, extrude discs have a narrow collum, and they are broader than protrude disc. Extrude discs can exhibit elongation to the above or under of the level of them. Hernia discs that are
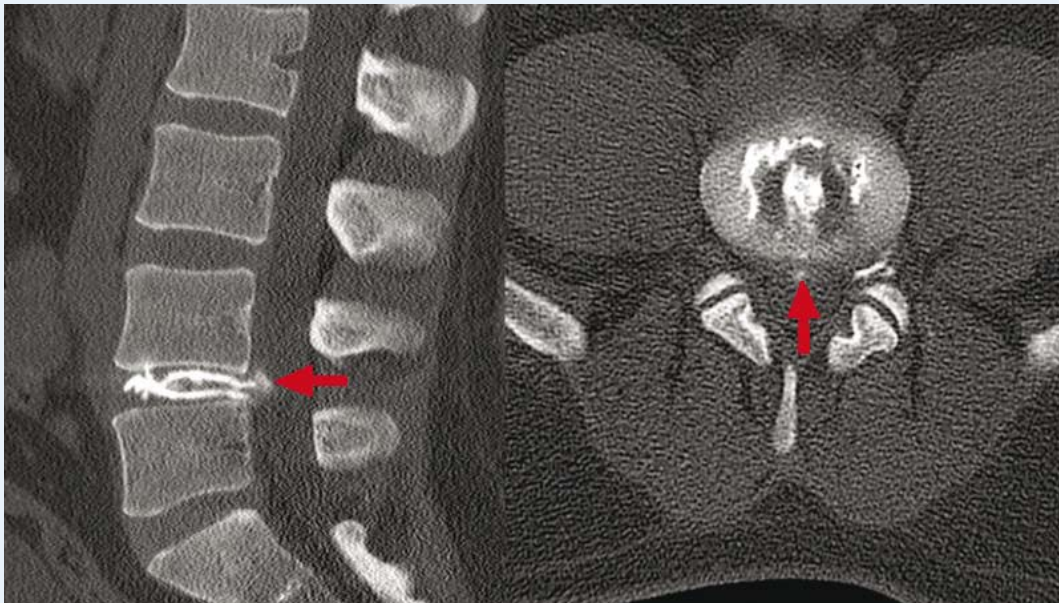

Figure 8: Dallas discogram, fifth degree annular tear. In axial CT profile and reformatted sagittal image, it can be seen that the contrast agent injected to the central region of intervertebral disc spread to the out of the disc by passing through the tear located in outer annular layer.
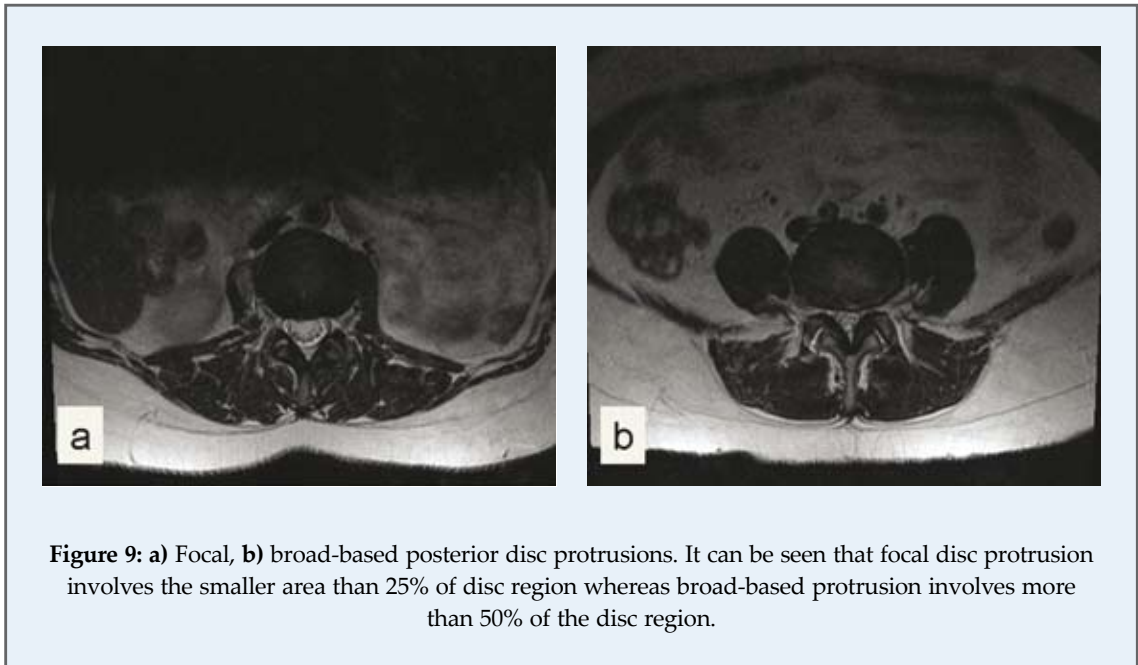
Figure 9: a) Focal, **b)** broad-based posterior disc protrusions. It can be seen that focal disc protrusion involves the smaller area than 25% of disc region whereas broad-based protrusion involves more than 50% of the disc region.

completely disconnected from their originating disc can be named "sequestered fragment". Sequestered fragment can be a contraindication for the minimal invasive treatments such that microdiskectomy, percutaneous radiofrequency ablation, percutaneous mechanic disc compression, intradiscal steroid injection, and so on [29]. It is not obligatory but optional to make distinction between extrude and protrude disc, and some radiologists and clinicians use the more extensive notion "herniation" in each case. Herniations can be subclassified to two subgroups that are "contained" and "uncontained" with respect to whether outer annulus that shields the disc herniation is intact, or not.

However, by non-invasive techniques such as CT or MR imagining, this distinction can not be made in every case. Even though discography is enough to make this distinction, it can constitute illusive images proceeded from posterior longitudinal ligament (PLL) that encloses the extrude disc, and/or from peridural membrane ("contained") (Diskografi bu ayrımı yapmada başarılı olsa da, ekstrüde diski çevreleyen posterior longitudinal ligaman (PLL) ve/veya peridural membrane devamlı ("contained") yalancı görünüm oluşturabilmektedir). Disc herniations can be also subclassified with respect to its relationship with PLL. Herniations bounded in PLL are classified as "subligamenteous herniation", whereas herniations that elongate to posterior by passing through PLL

are classified as "transligamentous herniation". On the other hand, disc herniations that locate at the out of PLL's region are classified as "extraligamentous herniation" (Figure 10).

However, it usually can not be possible to distinguish PLL from outer fibers of annulus or dural sac. The localization of disc herniations is specified with respect to its relationship between the anatomic structures such that facet joint, pedicul, and neural foramen in the axial, coronal, and sagittal images. The regions used in axial sections can be named as "central", "subarticular", "foraminal", "extraforaminal", or "distant lateral", and "anterior". On the other hand, in coronal and sagittal plans, the elongation of herniation can be named as "suprapedicular", "pedicular", "infrapedicular", and "disc plane". The big portion of disc herniations locates more than one region. The mostly attacked regions are the central and subarticular regions with the frequency rate reaching to 90%. The disc herniations placed at this localization affects the nerve root that rises from dural sac at that level. Only the 4-5% of the disc herniations locate foraminal or extraforaminal, and the herniations placed at this localization generally affects the roots that rise from upper level. The amount of hernia fragment is described regarding of the decrease of column diameter caused by this herniation. The measurements are made where the column has the narrowest column.
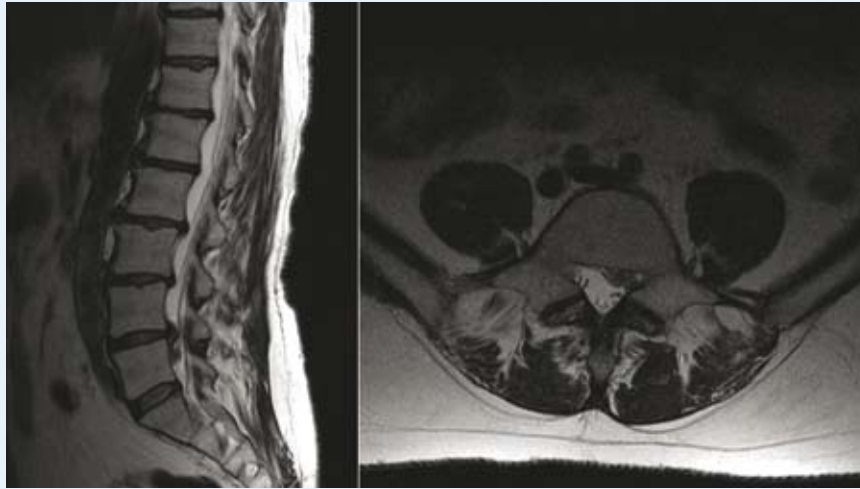
Figure 10: Caudal displacive herniation; in the sagittal and axial T2 weighted images, they are seen the left root compression, and the extrude herniation that caudally extends over left paracentral pedicular plane within the range of L4-L5.

If the column diameter constricts less than the ratio of 1/3, it is called "mild", if it constricts within the range of 1/3 and 2/3, it is called "moderate", and if it constricts more than the ratio of 2/3, it is called "severe". However, if this description is not correlated with clinical symptoms, it is not meaningful.

3. Degenerative Disc Variations in Posterior Elements

The functions of intervertebral disc and facet joints are closely related with each other. Degenerative alterations including the posterior elements affect the apophyseal joints, neural arcus bone structures, soft tissue plans located intermediate and posterior ligaments. By radiology and clinics, although it is given more importance to intervertebral disc alterations, the considerable portion of back ache is caused by the degeneration of posterior elements. Facet joints consists of superior and inferior articular process located reciprocal, coated with cartilage, and that joints are enclosed by synovium. Facet joints are located at the servical region in the sagittal plan; on the other hand, they are located throcal and lumbar region in the oblique and coronal plan. The joint is bounded by a thick capsule at the back.

In front, however, there is ligamentum flavum instead of capsule. Synovium elongates at the adjacency of flavum in front. Facet osteoarthritis emerges after mid-thirties, and it can be seen in all patient and generally exhibit asymptomatic feature after mid-sixties. Although the patients with degenerate facet generally complain about axial ache, radiculopathy and myelopathy are encountered symptoms as well. The recurrent stress and trauma related with overload is accused as stimulating factor. The first symptoms of degeneration are the fibrillation in the articular cartilage and erosions, and it progresses with the stenosis of joint spacing, sclerosis, and the formations of osteophyte. In some patients, it can be seen the subluxation at the joint, and comorbidit soft tissue alterations. Continuing stress causes the hypertrophy at the joint [30]. Pathria et al. [31] classified the facet joint arthrosis in four groups as it is seen below.

Stage 0: Normal.

Stage I: The slightly stricture in facet joint spacing and the irregularity at the surfaces of joint.

Stage II: The moderately stricture in facet joint spacing, the irregularity at the surfaces of joint, sclerosis, and hypertrophy.

Stage III: The severe degenerative alterations with significant stricture in facet joint spacing, sclerosis, and osteophytes.

CT imagining is more successful than MR imagining in detection of the early stage facet arthrosis. In CT imagining, the typical symptoms of facet arthrosis are the formations of osteophytes, subcondral sclerosis, formations of cyst, and stenosis of joint spacing (Figure 11). In addition to MR imagining, it can be seen the signal density uyumlu with edema at adjacent soft tissue plans and at posterior elements (MR
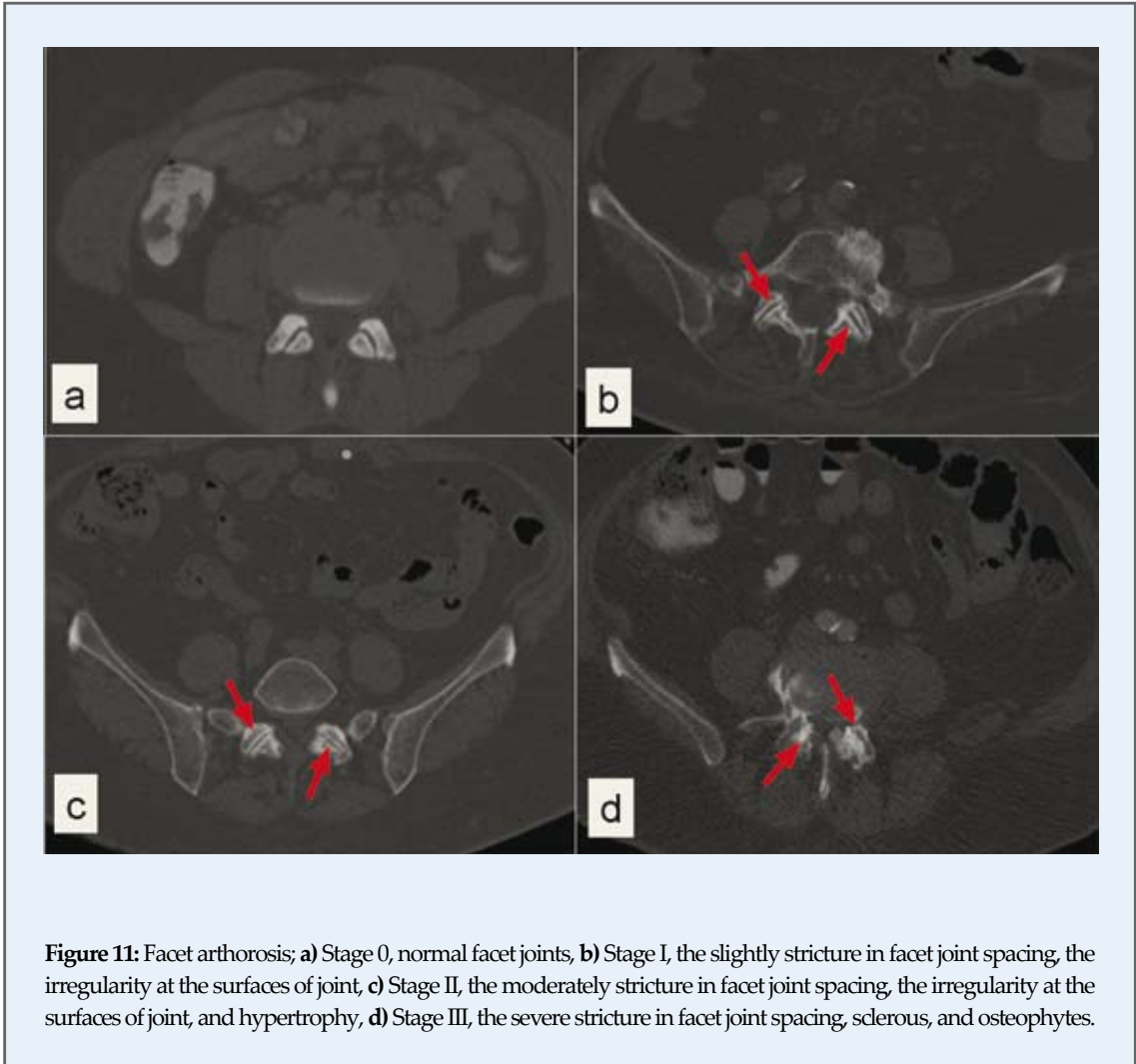
Figure 11: Facet arthorosis; **a)** Stage 0, normal facet joints, **b)** Stage I, the slightly stricture in facet joint spacing, the irregularity at the surfaces of joint, **c)** Stage II, the moderately stricture in facet joint spacing, the irregularity at the surfaces of joint, and hypertrophy, **d)** Stage III, the severe stricture in facet joint spacing, sclerous, and osteophytes.

görüntülemeye ek olarak posterior elemanlarda ve komşu yumuşak doku planlarında ödem ile uyumlu sinyal yoğunlu izlenebilmektedir).

It can be seen frequently the cystic lesions at the adjacency of facet joint. These cystic lesions are synovial cysts, ganglion and ligamentum cysts. Real synovial cysts are enclosed with synovial tissue, and they are directly related with joint spacing. The ganglion cysts, which do not have these features, include myxoid material. It is not possible to discriminate these two cysts with radiology.

The synovial cysts most frequently originate from lumbar region in the spinal column, and generally they locate at the level of fourth and fifth lumbar (L4-L5) vertebral that is more unstable than other segment. Rarely, they can be seen at the level of second and third (L2-L3) vertebral, or at the level

of fifth lumbar and first sacral (L5-S1) vertebral as well. Cervical and thoracal spinal column is attacked less frequently. Facet cysts located anterior can cause the compression of neural element by elongating to central canal or neural foramen with respect to where they place.

On the other hand, posterior facet cysts, which are seen more frequently than anterior facet cyst, are generally asymptomatic. By MR of CT imagining, facet cysts are detected as the pedicule defect, or well-bounded extradural located lesions that place at the adjacency of degenerate facet (Figure 12). In the cyst, they can be seen the hemorrhage, gas, mural calcification, and peripheral contrast involvement that is clearer in MR imagining.

It is thought that the main factor stimulating facet arthrosis is disc degeneration. Biomechanic studies
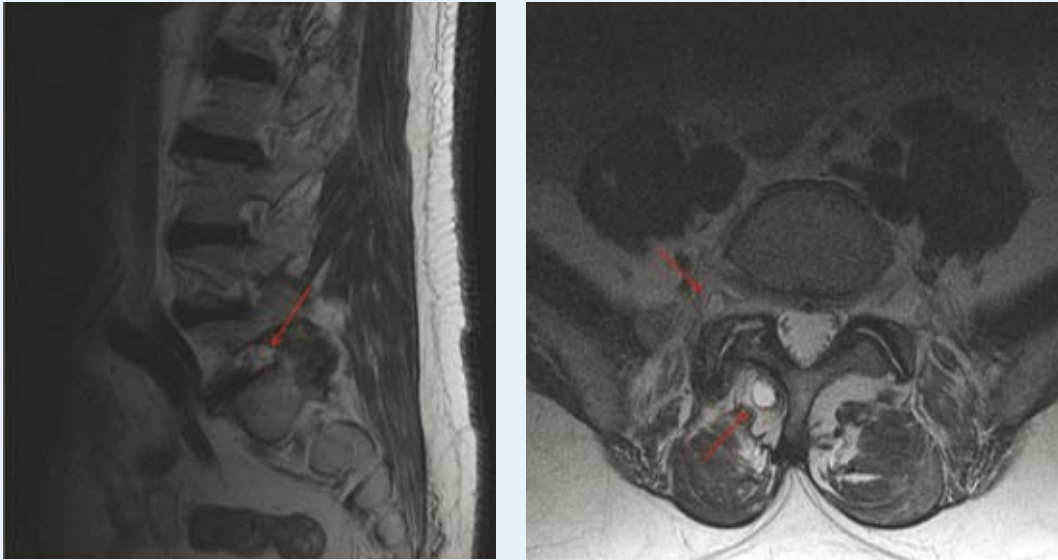
Figure 12: In the sagittal and axial images, the synovial cyst arising from L5-S1 right facet joint erases the right L5 root perineural tissue plans in the foramen. Another synovial cyst is seen at the posterior region of right facet joint.

have showed us that the stenosis in disc aperture causes a significant increase in load on facet joints [32]. It is accepted that segmental instability, which is seen in early degeneration, is also one of the causes preparing facet anthrosis.

Facet joints, which exhibit facet tropism ("asymmetric facet") and more sagittal orientation than normal, are also found guilty in arthrosis etiology [33]. However, it is currently debated point that these changes are the causes of facet arthrosis, or outcome of it.

The stenosis in intervertebral disc aperture causes the contact of spinous process, and as an outcome of this situation, degeneration at spinous process, and interspinous ligament. Interspinous pseudoarthrosis progresses to the cyst formations, redundant interspinous ligament, and as an outcome of these, interspinous located bursitis that is also named as "Baastrup phenomenon" [34]. Baastrup phenomenon causes to the stenosis in canal diameter, and local sensitivity; and it can be treated with local steroid injection [35] (Figure 13).

Figure 13: Baastrup phenomenon; in sagittal image, they are seen the bursitis in L5-S1 interspinous aperture, and cyst that elongates to posterior epidural region and causes to the compression of sac.

Lumbar Degenerative Disc Disease and Dynamic Stabilization

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