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Osteoarthritis of the spine: the facet joints

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Abstract

Osteoarthritis (OA) of the spine involves the facet joints, which are located in the posterior aspect of the vertebral column and, in humans, are the only true synovial joints between adjacent spinal levels. Facet joint osteoarthritis (FJ OA) is widely prevalent in older adults, and is thought to be a common cause of back and neck pain. The prevalence of facet-mediated pain in clinical populations increases with increasing age, suggesting that FJ OA might have a particularly important role in older adults with spinal pain. Nevertheless, to date FJ OA has received far less study than other important OA phenotypes such as knee OA, and other features of spine pathoanatomy such as degenerative disc disease. This Review presents the current state of knowledge of FJ OA, including relevant anatomy, biomechanics, epidemiology, and clinical manifestations. We present the view that the modern concept of FJ OA is consonant with the concept of OA as a failure of the whole joint, and not simply of facet joint cartilage.

Introduction

Osteoarthritis (OA) of the spine involves the facet joints, otherwise known as the zygapophyseal joints. These paired diarthrodial joints in the posterior aspect of the vertebral column are the only true synovial joints between adjacent spinal levels in humans. Facet joint osteoarthritis (FJ OA) is intimately linked to the distinct but functionally related condition of degenerative disc disease, which affects structures in the anterior aspect of the vertebral column. FJ OA and degenerative disc disease are both thought to be common

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causes of back and neck pain, which in turn have an enormous impact on the health-care systems and economies of developed countries.[1–4]

FJ OA has to date received far less critical study than other important OA phenotypes such as knee OA. Over the past two decades our conceptualization of knee OA has shifted away from a predominant focus on cartilage degeneration towards a view of OA as a heterogeneous and dynamic process of whole-joint failure resulting from an imbalance between the breakdown and repair of joint tissues.[5] This perspective of OA has not yet been applied to the facet joints. This Review presents current data on FJ OA, including relevant anatomy, biomechanics, epidemiology, and clinical manifestations. Treatments for facet joint pain are outside the scope of this Review and are covered elsewhere.[6] We present the view that the modern concept of FJ OA, as linked to other degenerative conditions of the spine such as degenerative disc disease, is in harmony with the contemporary conceptual model of OA as a failure of the whole joint and not simply of facet joint cartilage.

Definition of FJ OA

FJ OA is a clinical and pathological construct that involves the functional failure of the synovial facet joints. Although often viewed as a disease of articular cartilage loss and bony hypertrophy, the process of failure actually involves the whole joint, including the subchondral bone, cartilage, ligaments, capsule, synovium, and periarticular paraspinal muscles and soft tissues. The facet joint itself is a component of a spinal ‘motion segment’ (Figure 1) that also includes the intervertebral disc, which tends to degenerate in concert with the facet joint. As such, FJ OA is frequently associated with degenerative disc disease.[1–5,7–11]

Imaging

The classic radiographic hallmarks of FJ OA involve both degenerative and proliferative features, including narrowing of the facet joint space, subarticular bone erosions, subchondral cysts, osteophyte formation, and hypertrophy of the articular process.[12] Radiographic criteria for assessing these features were initially developed using plain radiographs, and have subsequently been adapted to CT and MRI.[13–15] Cross-sectional imaging with CT or MRI is required for a detailed evaluation of facet joint morphology in multiple planes, owing to the obliquity of the joints. Although CT is more accurate than MRI for distinguishing bony pathology, agreement between CT and MRI is moderate-to-good,[14] and MRI enables better resolution of non-bony pathology. Commonly used radiographic grading systems exist only for cervical and lumbar FJ OA.[12,14–16] These systems grade FJ OA using ordinal scales based on combinations of radiographic features (Table 1), with higher grades representing greater OA severity. No grading scales for evaluation of cervical or thoracic FJ OA using CT or MRI are in common usage.[16]

In addition to the classic radiographic features of FJ OA, more recent imaging techniques have utilized fat-suppressed MRI sequences, which are more fluid-sensitive than conventional MRI; use of these sequences has led to an improved ability to evaluate changes in the facet joints and surrounding structures. Subchondral bone marrow oedema-like lesions (BMLs), which are a frequent finding in knee OA and have been shown to be associated

with clinical manifestations of OA such as pain and structural progression,[1–3,5,12,14,16–21] are also seen in the facet joints. Recent studies using fat-suppressed MRI sequences in the spine have demonstrated that BMLs are present in the lumbar facet joint articular processes of between 14% and 41% of patients with back pain.[7–11,22–24] BMLs are also commonly seen in the cervical spine in routine clinical practice. Preliminary studies suggest associations between facet joint BMLs and the presence and location of back pain.[12,22,23,25] Additional features associated with FJ OA can also be detected using fat-suppressed MRI sequences, including facet joint effusions, interspinous ligament oedema, and periarticular oedema.[12,24] Importantly, these facet joint features seen on fat-suppressed MRI sequences are not included in the scales currently used for evaluation and grading of FJ OA (Table 1).

Topographically, FJ OA is not distributed evenly throughout the spine. In the lumbar region, the classic radiographic features of FJ OA are most common at the lower levels: FJ OA prevalence is highest at L4–L5, followed by L5–S1.[1,10,12–14] Other facet joint features including BMLs, effusions, and periarticular oedema are also most common at L4–L5 and L5–S1.[25] By contrast, in the cervical spine, based on cadaveric studies FJ OA seems to be more common in the mid-cervical region, at C3–C5;[26–28] however, no population-based imaging studies have corroborated these findings.

Features of FJ OA on imaging can overlap with those of inflammatory conditions that might affect the facet joints. FJ OA can be distinguished from these other conditions by a number of radiographic differences (Box 1).

Anatomy and biomechanics

The facet joints of the posterior spine are true synovial joints, anatomically and functionally distinct from the fibrocartilaginous articulation of the intervertebral disc. At every spinal level except C1–C2, the so-called ‘three-joint complex’, or motion segment, is formed by the three articulations between adjacent vertebrae: one disc and two facet joints (Figure 1). The spine can therefore be considered as a structure composed of multiple motion segments connected in series, with its total motion a composite of motion in the individual segments. At every level, the superior articular processes of the lower vertebra extend upwards and articulate with the smaller inferior articular processes of the vertebra above it. Joint alignment and load distribution are thought to be major factors in the development and progression of FJ OA, just as they are commonly implicated in knee OA.[16,29]

In the cervical and thoracic spine, the joint surfaces are roughly planar, but not truly flat, being reciprocally convex and concave; in the lumbar region, the facet joints are less planar and appear roughly boomerang-shaped when viewed from above. The cervical facet articular surface area is about two-thirds the size of the area of the vertebral end plate but in the lumbar region the facets are relatively smaller compared with the vertebral bodies. The facet joint exhibits features typical of synovial joints: articular cartilage covers the apposed surfaces of each of the facets, resting on a thickened layer of subchondral bone, and a synovial membrane bridges the margins of the cartilaginous portions of the joint. A superior and inferior capsular pouch, filled with fat, is formed at the poles of the joint, and a baggy fibrous joint capsule covers the joint like a hood. A fibroadipose meniscoid projects into the

superior and inferior aspect of the joint and consists of a fold of synovium that encloses fat, collagen, and blood vessels.[30] These meniscoids serve to increase the contact surface area when the facets are brought into contact with one another during motion, and slide during flexion of the joint to cover articular surfaces exposed by this movement.

The orientation of the facet joints varies by location within the spine, which relates to the principal planes of motion allowed at each spinal level. In the cervical region, the facet joints are inclined approximately 45° from horizontal. In the thoracic spine, the joints are oriented approximately 60° from horizontal and also are rotated roughly 20° in the axial plane, which tends to enable more axial rotation to occur at these levels. In the lumbar region, the facet joints are inclined to a nearly vertical orientation, and are curvilinear,[31] a shape that highlights their role in preventing rotation as well as forward displacement; an average of only 8° rotation is allowed in the lumbar spine, limited primarily by the facet joints.[31–33]

Associated spinal degeneration

Functionally, the three joints in each motion segment are highly interdependent, such that changes in one affect the other two and vice versa.[8,9] Thus, lesions that affect the disc tend to eventually have an effect on the facet joints, and trauma or instability of the posterior structures may in turn affect the disc. The motion segment has been best studied in the lumbar spine. In the majority of individuals, pathology begins in the disc and is followed by changes in the facet joints.[7,10,11] In the setting of a dysfunctional or unstable segment, the interplay between these three joints ultimately results in failure of the combined three-joint complex.[7] Later, as a result of biomechanical changes at this one level, pathological changes can occur in the motion segment at spinal levels above or below.[34] In support of this concept of disc and facet interdependence, FJ OA in the lumbar spine occurs at the levels most commonly affected by disc degeneration (L4–S1).[35] Both FJ OA and disc degeneration decrease in prevalence in the upper lumbar regions.

A complex relationship exists in the load-sharing between the facet joints and the disc, and depends largely on spinal posture. In most cases, the disc is the primary load-bearing structure in each motion segment. Biomechanical models of isolated spinal segments without disc degeneration or FJ OA have demonstrated that the load carried by the facets can be up to 33% of the total load borne by that spinal segment, and that more load is transmitted through the facet joints when the spine is extended and less when it is flexed or in a neutral position.[36] Degenerative disc disease and disc-space narrowing cause a marked increase in transmission of force across the facet joints, as less body weight is supported by the disc when it becomes functionally incompetent.[36] Up to 70% of an axial load can be borne by the facet joints in cases of severe disc-space narrowing.[37]

The roles of the spinal musculature are twofold. The first role is to control the movement of the spine and to contribute essential stabilization to the vertebral column, which would buckle without muscular support.[38,39] The second role is to provide proprioceptive feedback regarding the position of the spine in space.[39,40] With increasing age, paraspinal muscle mass decreases,[41] which could compromise both of these important functions and contribute

to FJ OA by allowing poorly controlled segmental motion. In support of this concept, patients with chronic low back pain seem to have both impaired spinal proprioception[42] and a decrease in paraspinal muscle cross-sectional area.[43] A recent population-based study demonstrated an association between decreased paraspinal muscle density and the presence of lumbar FJ OA.[41]

Adult degenerative scoliosis (spinal deformity or curvature in the coronal plane) and degenerative spondylolisthesis (displacement of one vertebra relative to another in the sagittal plane) are also thought to be related to FJ OA[44,45] and failure of the motion segment.[46,47] In degenerative scoliosis, a cycle of asymmetric deformity, asymmetric loading, and asymmetric degeneration occurs, with progressive scoliotic deformity leading to still further increased force transmission through the facet joint on the concave side of the curve.[46] In degenerative spondylolisthesis, subluxation of the facet joint occurs, related to progressive loss of cartilage and articular remodeling as part of OA. Facet joints at spinal levels affected by degenerative spondylolisthesis have been found to be more sagittally oriented than those at levels without spondylolisthesis.[15,48] A more sagittal joint orientation might lessen the amount of anterior restraint that the facet joints are able to supply to the vertebral column simply because there is less of a bony barrier in the sagittal plane. This lack of restraint can result in anterior slippage of the superior vertebra in the motion segment. Spondylolisthesis most often occurs at L4–L5, the same level that is most often affected by FJ OA.[10,15]

Histologic and macroscopic changes

The earliest changes of FJ OA involve the articular cartilage, synovium, and capsule. In later stages, the subchondral bone and bony joint margins are affected (Figure 2).[49] Changes to cartilage begin with fibrillation and shallow pitting that affect the cartilage surface focally, followed by deeper fibrillation and fissuring, flaking and pitting, then erosion down to subchondral bone.[50] Joint-space narrowing results from progressive cartilage thinning.[51] Chondrocyte clusters can be seen in early cartilage lesions, with the presence of foci of fibrocartilage suggesting attempts at repair.[52] Early on, the joint capsule might show fibrosis and increased vascularization, with the presence of inflammatory cells.[53] Later, progression to extensive fibrocartilage proliferation occurs throughout the hypertrophied posterior capsule, and is especially pronounced at the capsular attachment.[54]

Osteophyte formation and remodelling of the subchondral bone are the most conspicuous remodelling phenomena in FJ OA, but these occur in the later stages of the pathology, and might be absent even in cases of severe cartilage damage.[52] When present, osteophytes are located mostly at the lateral margins of the joint at the site of the capsular insertion. In addition to subchondral thickening and sclerosis, subchondral cysts can be seen, similar to those in the subchondral bone of appendicular joints affected by OA.

Over the course of adult life, the superior and inferior facets of a typical lumbar vertebra exhibit different patterns of changes, reflecting the different stresses placed upon these parts of the joint. In animal models of FJ OA and in gross histologic studies in humans, areas of early degenerative change occur in focal locations of the joint, suggesting that damage

occurs in regions that experience the greatest mechanical forces.[30] The cartilage in the central regions of the facet joints is relatively spared, with the majority of changes occurring at the superior and inferior poles where the articular processes contact one another during flexion and extension.[49,50,53,55]

Degenerative changes in facet joint cartilage can begin at an early age, with a recent gross histologic report in a population of organ donors finding fibrillation and flaking of the articular cartilage as early as age 15 years.[50] In elderly adults, degenerative facet joint cartilage lesions were found on gross histologic examination in 80% of individuals, with most found at the L4–L5 level; in this same population, proliferative features of OA including osteophytosis were less common and found in only 33% of individuals. The joints that did show osteophyte formation had more advanced degeneration than those without osteophytes.[30]

Prevalence and progression

Determining the population-based distribution and determinants of FJ OA is hindered by the fact that almost all studies to date have included selected samples of cadavers or clinical patients. Nevertheless, the few population-based estimates available make it evident that FJ OA is widely prevalent in adults. Although no studies of cervical FJ OA prevalence have used advanced imaging, cervical FJ OA (defined as Kellgren–Lawrence grade [KLG] ≥2 on lateral radiographs) was prevalent in 19% of adults age 45–64 years, and in 57% of adults age 65 years and older, in a community-based US population;[56] these estimates probably underestimate true prevalence because of the insensitivity of radiography. No population-based studies of thoracic FJ OA have been performed. Amongst community-based adults in the Framingham Heart Study, moderate or severe lumbar FJ OA on CT imaging was present in 36% of adults age <45 years, 67% of adults age 45–64 years, and 89% of those age 65 years and older.[7] No studies have assessed incident FJ OA in any spinal region, but amongst community-based US adults with cervical FJ OA KLG ≥2 at baseline, the overall rate of radiographic progression (defined as an increase of ≥1 KLG) was 8 cases per 100 person-years of observation.[57]

Risk factors and correlates

Many risk factors for FJ OA have been proposed, including age, sex, overweight, physical trauma, occupational factors, smoking, and others. However, few studies have examined risk factors in unselected population-based samples, and nearly all studies have been cross-sectional.

In the cervical region, studies of risk factors for FJ OA have used plain radiography. Age is strongly associated with prevalent cervical FJ OA,[56,58] as well as with its progression.[57] There seems to be no association of gender with prevalent cervical FJ OA, but men are slightly more likely to have FJ OA progression.[59] Higher BMI is associated with greater prevalence of cervical FJ OA in women (adjusted OR 1.02 per BMI unit; 95% CI 1.00–1.03) and men (adjusted OR 1.03; 95% CI 0.99–1.06).[58] The magnitude of this association of cervical FJ OA with BMI, however, is quite small: as an example, in a woman of roughly average height (1.62 m), an increase in weight of 2.62 kg (equivalent to one BMI unit)

would confer only 2% greater risk of having cervical FJ OA. Occupational factors such as stair-climbing, standing, and jolting activities are not associated with cervical FJ OA, but other potentially relevant factors—including lifting, pulling, and carrying—have not been examined.[58]

In the lumbar region, age is strongly associated with prevalent FJ OA.[56,60] Women are more likely than men to have FJ OA on lumbar CT (adjusted OR 1.86; 95% CI 1.09–3.18)7 and plain radiography (OR 1.52; 95% CI 1.14–2.0).[61] African Americans have a substantially lower risk of FJ OA on lumbar radiographs as compared with white Americans (OR 0.42; 95% CI 0.31–0.56).[61] High BMI is also independently associated with lumbar FJ OA,[7,61] and the magnitude of the association is much larger than with cervical FJ OA: the risk of FJ OA on lumbar CT is almost threefold higher in overweight individuals (BMI 25–30 kg/m2), and fivefold higher in obese individuals (BMI 30–35 kg/m2), when compared with a normal-weight reference group (BMI ≤25 kg/m2).[60] Abdominal aortic calcifications are associated with lumbar FJ OA, but smoking and other cardiovascular risk factors are not.[60] In addition to these traditional epidemiologic risk factors, some studies have examined associations between specific characteristics of spinal anatomy and lumbar FJ OA. Concurrent disc-height narrowing is associated with roughly doubled odds of FJ OA, independent of age, sex, and BMI.[7] A more sagittal orientation of the facet joints (as opposed to a coronal orientation) is associated with a higher prevalence of FJ OA at the L4–L5 spinal level, but facet joint asymmetry, or 'tropism', is not associated with lumbar FJ OA.[15] Poor quality of extensor muscle has also been linked to FJ OA at the L4–L5 spinal level.[41] It should be noted that the cross-sectional design of the studies from which these results were obtained means that spinal anatomy risk factors identified could indicate either causes or consequences of FJ OA, and studies of incidence and progression are lacking.

Hereditary factors seem to be major determinants of intervertebral disc degeneration,[62] and genetic influences could also explain variation in prevalence or severity of FJ OA. Hand, knee, and hip OA all have a substantial genetic component and frequently coexist,[63] but, to date, no studies have directly examined the role of heredity in FJ OA.

Clinical manifestations

Although low back pain (LBP) and neck pain represent complex disorders that are best addressed within the framework of a biopsychosocial model, a clear evaluation of the part that FJ OA plays in this context has been largely neglected. About half of adults suffer from neck pain and two-thirds from LBP at some point in their lifetime.[3,64] Although intervertebral disc degeneration is the pathoanatomic finding most frequently linked to spinal pain in younger adults, FJ OA might have greater importance as a cause of pain in older adults. Nevertheless, FJ OA represents only one of many potential sources of spinal pain in older adults, where pain can also be caused by disc degeneration or the paraspinal musculature and ligaments, and less commonly by vertebral fractures, spinal infections, neoplasms, rheumatoid arthritis, seronegative spondyloarthritis, and other conditions. As with other types of degenerative spine pathology, such as disc degeneration, the imaging finding of FJ OA is neither highly specific for spinal pain, nor specific for identifying the anatomic source of pain when spinal pain is present. Nonetheless, it is now well established

that facet joints can be a source of spinal pain, although even this assertion was a topic of debate for many years.[65]

Pain from osteoarthritic joints probably derives from a number of factors. Facet joint cartilage is aneural, and pain associated with FJ OA can arise from nociceptors within and surrounding the joints, including nociceptors in the bone itself. The facet joints and their capsules are well innervated by the medial branches of the dorsal primary rami of the spinal nerves, where both free nerve endings and mechanoreceptors have been identified.[66] The capacity of facet joints to produce pain has been demonstrated in normal volunteers through injection of irritant solutions into both the cervical and lumbar spine,[67,68] and has also been established by the analgesic effect of anaesthetic injected into the joints themselves.[17] Mechanical factors that could activate nociceptor fibres include direct pressure on subchondral bone, intramedullary hypertension, trabecular microfractures, capsular distension, and synovial inflammation, all of which could secondarily result in reflex muscular spasm of the erector spinae, multifidi, and other paraspinal muscles.[69] Prolonged peripheral inflammation in and around facet joints can lead to central sensitization, neuronal plasticity, and the development of chronic spinal pain.[6,70]

The clinical syndrome related to painful facet joints is usually localized neck pain or back pain with some degree of radiation into the upper or lower limbs. Radiation from painful mid-cervical and lower-cervical facet joints tends to produce pain in the posterior scapular region with some radiation around the shoulder girdle, whereas painful upper cervical facet joints tend to refer pain to the posterior occipital region and can produce headache (Figure 3a).[68,71] In the lumbar spine, referred pain is predominantly in the buttock and the thigh; radiation past the knee is rare.[71] Lumbar facet joint pain referral maps vary substantially between different studies;[72–75] Figure 3b presents a composite map of lumbar facet joint referred pain. Referred pain from the facet joints needs to be differentiated from true radicular pain, which is caused by nerve root compression or irritation. Radicular pain tends to travel farther distally in the limb than lumbar facet joint pain, and can be associated with neurologic findings such as motor or sensory loss, or diminished reflexes.

Clinical examination in cases of suspected symptomatic FJ OA should focus on ruling out disc herniation or spinal stenosis with neurologic deficits, as well as more concerning, though rare, infectious, fracture-related, or neoplastic aetiologies. No examination maneuvers are pathognomonic for symptomatic FJ OA, and mechanical tests purported to stress the facet joints probably load the intervertebral discs and the ligaments as well, decreasing their potential predictive value. In routine clinical practice, back pain that worsens with lumbar hyperextension, extension–rotation, or straightening from flexion is often considered to be indicative of a symptomatic facet joint. However, these and other criteria have not been corroborated by investigations.[20,76] Other features, such as localized unilateral back pain, pain with palpation over the facet joint, pain relieved by flexion, and pain not radiating past the knee, are also commonly used clinically to diagnose a symptomatic facet joint.[77]

Epidemiological studies evaluating the association between radiographic FJ OA and pain have produced conflicting results,[1,2,78–80] with several studies showing no relationship

between the presence or severity of FJ OA on CT and/or MRI findings and the presence of LBP or neck pain.[81–83] Unfortunately, most imaging studies to date have not used reliable and well-developed scales for assessment of FJ OA. Owing to the lack of specificity of FJ OA on imaging, anaesthetic blockade using fluoroscopically guided injections has become standard practice for diagnosing whether a particular joint is producing pain. Many authors support the conjecture that anaesthetic blocks represent the best diagnostic test to confirm pain arising from the facet joints.[68,76,84,85] A working diagnosis of facet-mediated pain can theoretically be confirmed by injection of local anaesthetic either into the facet joint itself or around the medial branches of the dorsal rami that supply the sensory innervation to the joint. However, unacceptably high false-positive rates of 20–40% have been reported for such blocks performed without placebo or comparative anaesthetic controls,[86] leading to continuing controversy over the ideal diagnostic paradigm and the validity of diagnostic blocks.[87] Using controlled diagnostic anaesthetic blocks, however, the reported prevalence rates for lumbar facet-mediated pain ranges from 15% in a sample of injured workers to 52% in older adults with longstanding back pain seen in a pain clinic.[17,76,84,88] Figure 4 demonstrates a correlation between mean sample age and the prevalence of back pain attributable to the lumbar facet joints in studies using controlled diagnostic blocks. This trend mirrors the increasing prevalence of FJ OA with increasing age, and points to FJ OA as one possible explanation for the higher prevalence of facet-mediated pain seen in older adults as compared with younger adults.[88]

Besides its importance in producing spinal pain, FJ OA can have secondary effects on neighboring structures, namely the traversing spinal nerve roots. The osteophytosis and articular hypertrophy seen with FJ OA can result in narrowing (or ‘stenosis’) of the central spinal canal, the lateral recess, and the intervertebral foramina, with potential impingement on spinal nerve roots at these locations.89 Synovial cysts, which sometimes develop in association with FJ OA, can compound this stenosis. In addition, degenerative spondylolisthesis, which may result from instability of the three-joint complex, can contribute further to stenosis. In some individuals, stenosis can cause symptoms of radicular pain or neurogenic claudication, which are distinct from typical facet joint pain referral patterns.

Conclusions

FJ OA may best be defined as a disease of whole-joint failure, in which injury to one component of the spinal motion segment leads to damage of other components and, collectively, to segmental failure. This process of facet joint failure can be associated with back pain or neck pain. Similar to the modern concept of appendicular OA, local mechanical factors within the spinal motion segment can promote susceptibility to, and progression of, FJ OA. Alterations in the structural integrity of the three-joint complex, particularly the degeneration of the intervertebral disc, as well as abnormal joint alignment or loading and paraspinal muscular weakness are implicated as risk factors for development of FJ OA. Clinically, the prevalence of facet-mediated pain increases with increasing age, which suggests FJ OA might have an important role in the ageing population with spinal pain.

Review criteria

A review of the literature was performed by searching the PubMed database for relevant articles in English up to May 2012. The search terms used were “arthritis” or “osteoarthritis” in combination with “facet joint” or “zygapophyseal joint”. Titles were screened for relevance to the topic of this article, abstracts were reviewed where indicated, and relevant full articles were retrieved. Other articles were identified from the bibliographies of retrieved articles and the authors’ personal collections. The reference list was last updated in September 2012.

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Key points

- Osteoarthritis of the spine involves the facet joints, the only true synovial joints between adjacent spinal levels in humans
- The classic radiographic hallmarks of facet joint osteoarthritis (FJ OA) include narrowing of the facet joint space, subarticular bone erosions, subchondral cysts, osteophyte formation, and hypertrophy of the articular processes
- FJ OA is widely prevalent in older adults, and is thought to be a common cause of back and neck pain
- FJ OA may have a particularly important role in the ageing population with spinal pain
- As with osteoarthritis of the extremities, FJ OA can be more accurately viewed as a failure of the whole-joint organ, and not simply of facet joint cartilage

Box 1 | Differential diagnosis of FJ OA

Rheumatoid arthritis

RA is the most common inflammatory disorder affecting the spine, and has a predilection for the cervical region. Depending on the diagnostic criteria used, cervical facet involvement is seen in 25–80% of cases of RA.[93] Conversely, involvement of the lumbar or thoracic spine is rare, affecting only 3–5% of patients with RA.[94] Compared with OA, erosive features are more pronounced in RA, and these changes might be associated with ligamentous laxity, instability, and subluxations. By contrast, proliferative features are more prevalent in FJ OA than RA.[94,95]

Spondyloarthritis

Seronegative spondyloarthritis can also have features that overlap with FJ OA. Among subtypes of this condition, ankylosing spondylitis is the most common, and typically affects the sacroiliac joint, though the facet joints can also be involved. Unlike FJ OA, radiographic manifestations of ankylosing spondylitis include a ‘squaring’ of the vertebral bodies, ligamentous ossification, and development of bridging syndesmophytes.[93] The clinical manifestations of progressive loss of lordosis and involvement of costovertebral joints are more typical of ankylosing spondylitis than FJ OA. Psoriatic arthritis also affects the spine radiographically in up to 70% of cases,[96] more commonly affecting the cervical spine than other regions. Ligamentous ossification and syndesmophyte formation is often present; alternatively, erosive features may predominate with subluxation.

Gout

Gout occasionally involves the axial skeleton, but data are sparse on the prevalence of spinal involvement.[97] Tophaceous deposits in the facet joints might be identified on CT images.

Abbreviations: FJ OA, facet joint osteoarthritis; OA, osteoarthritis; RA, rheumatoid arthritis.

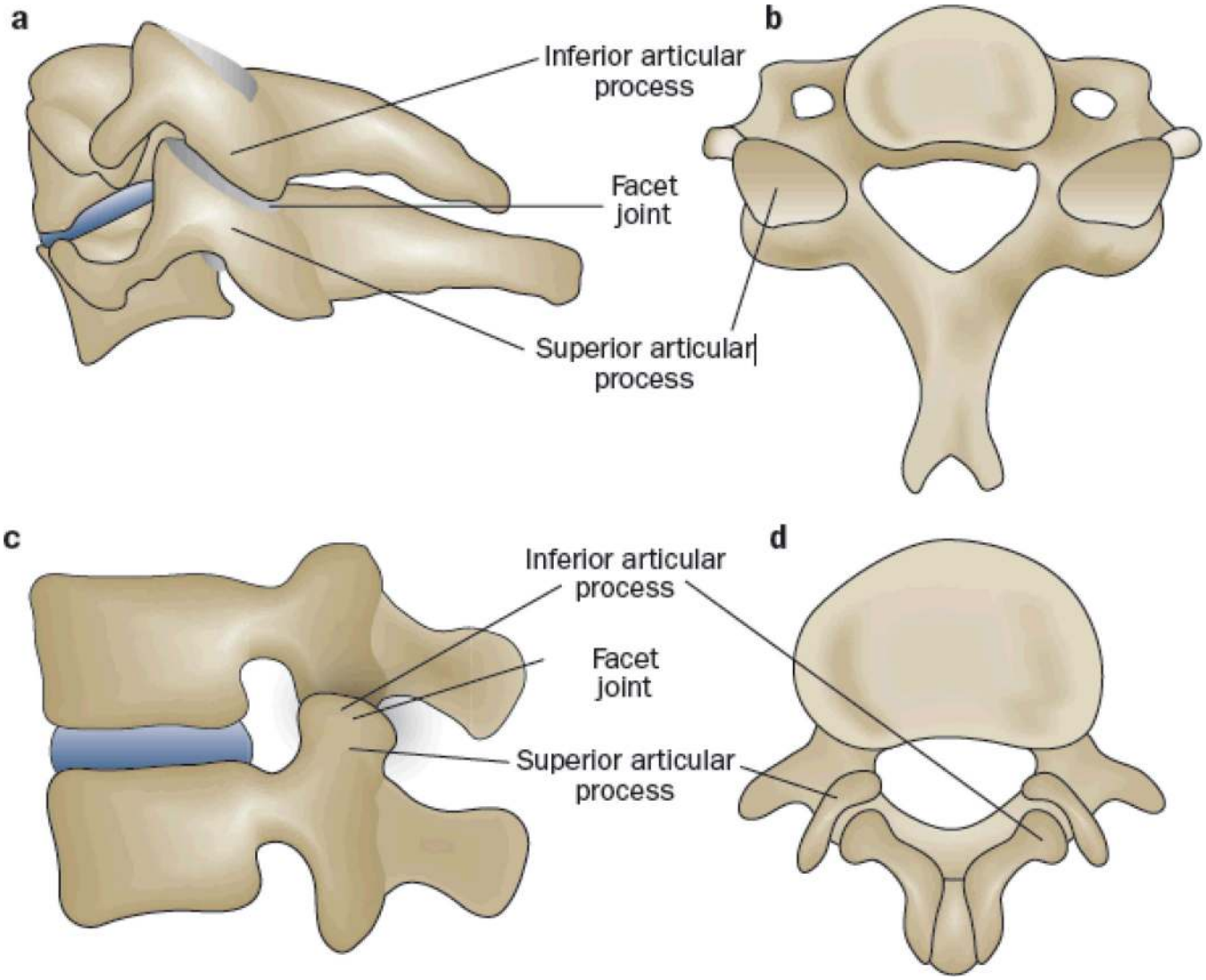

Figure 1.

Facet joint and intervertebral disc anatomy. At every spinal level, the paired facet joint and the intervertebral disc make up the 'three-joint complex', or the spinal 'motion segment'. **a** | Sagittal view of a cervical disc–facet unit. **b** | Axial view of a cervical disc–facet unit. The cervical facet joint space cannot be seen in cross-section owing to the orientation of the joint in the cervical region. **c** | Sagittal view of a lumbar disc–facet unit. The lumbar facet joint space cannot be seen in cross-section owing to the orientation of the joint in the lumbar region. **d** | Axial view of a lumbar disc–facet unit.

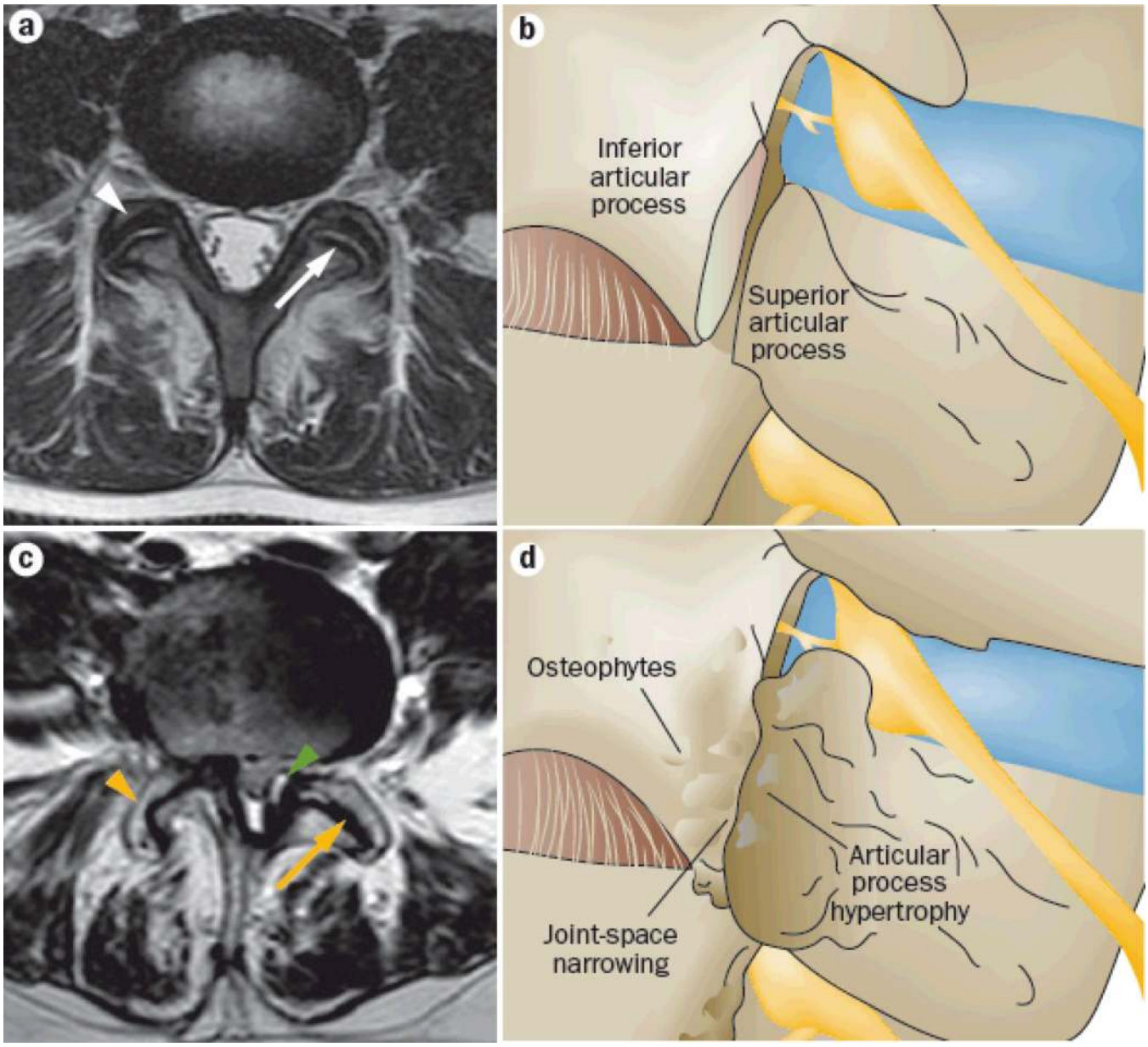

Figure 2.

Normal facet joints and advanced facet joint osteoarthritis. The intervertebral disc is shown in blue and nerves in yellow. **a** | T2-weighted axial MRI image of normal facet joints with no joint-space narrowing (white arrow), and no osteophytes or articular process hypertrophy (white arrowheads). **b** | Normal facet joints. **c** | T2-weighted axial MRI image of osteoarthritic facet joints with joint-space narrowing (yellow arrow), osteophytes and articular process hypertrophy (yellow arrowhead). Facet joint osteoarthritis, disc-bulging, and a facet joint synovial cyst (green arrowhead) in combination lead to stenosis of the central canal and lateral recesses. **d** | Osteoarthritic facet joints.

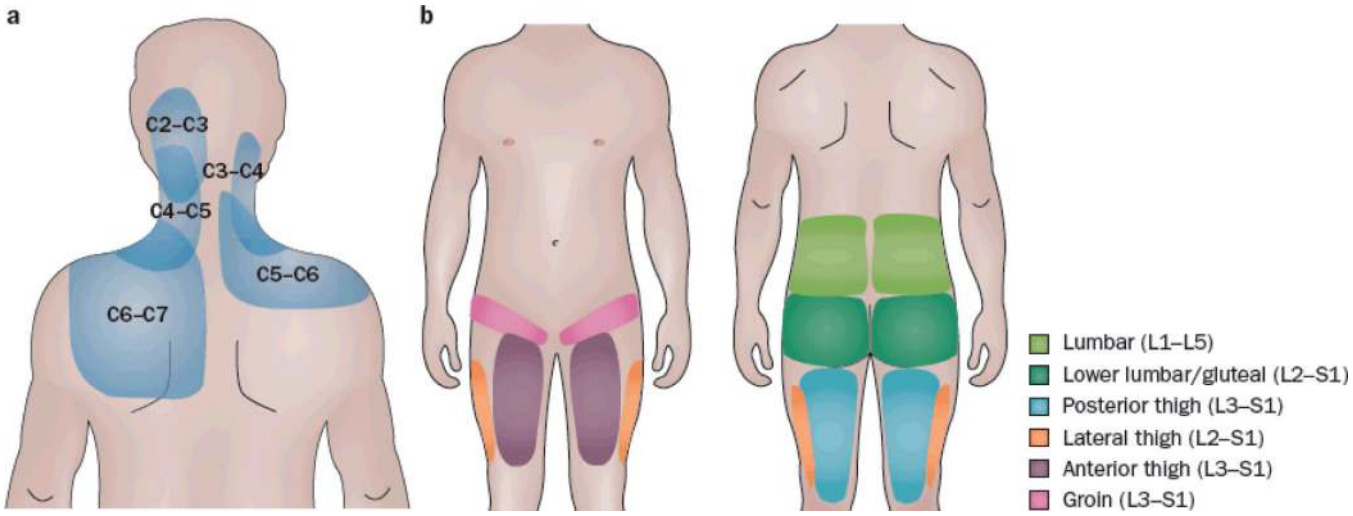

Figure 3.

Cervical and lumbar facet joint pain referral patterns. **a** | Distributions of pain referred from the cervical facet joints. **b** | Distributions of pain referred from the lumbar facet joints. This is a composite map of lumbar facet joint pain referral locations from multiple studies.[72–75] The lumbar and low lumbar/gluteal regions are the most common locations of pain. Although not depicted here, referral of pain distal to the knee can also occur, but is relatively rare. Part a adapted with permission from Lippincott Williams & Wilkins © Dwyer, A. *et al*. Cervical zygapophyseal joint pain patterns. I: A study in normal volunteers. *Spine (Phila Pa 1976)* 15, 453–457 (1990).[68]

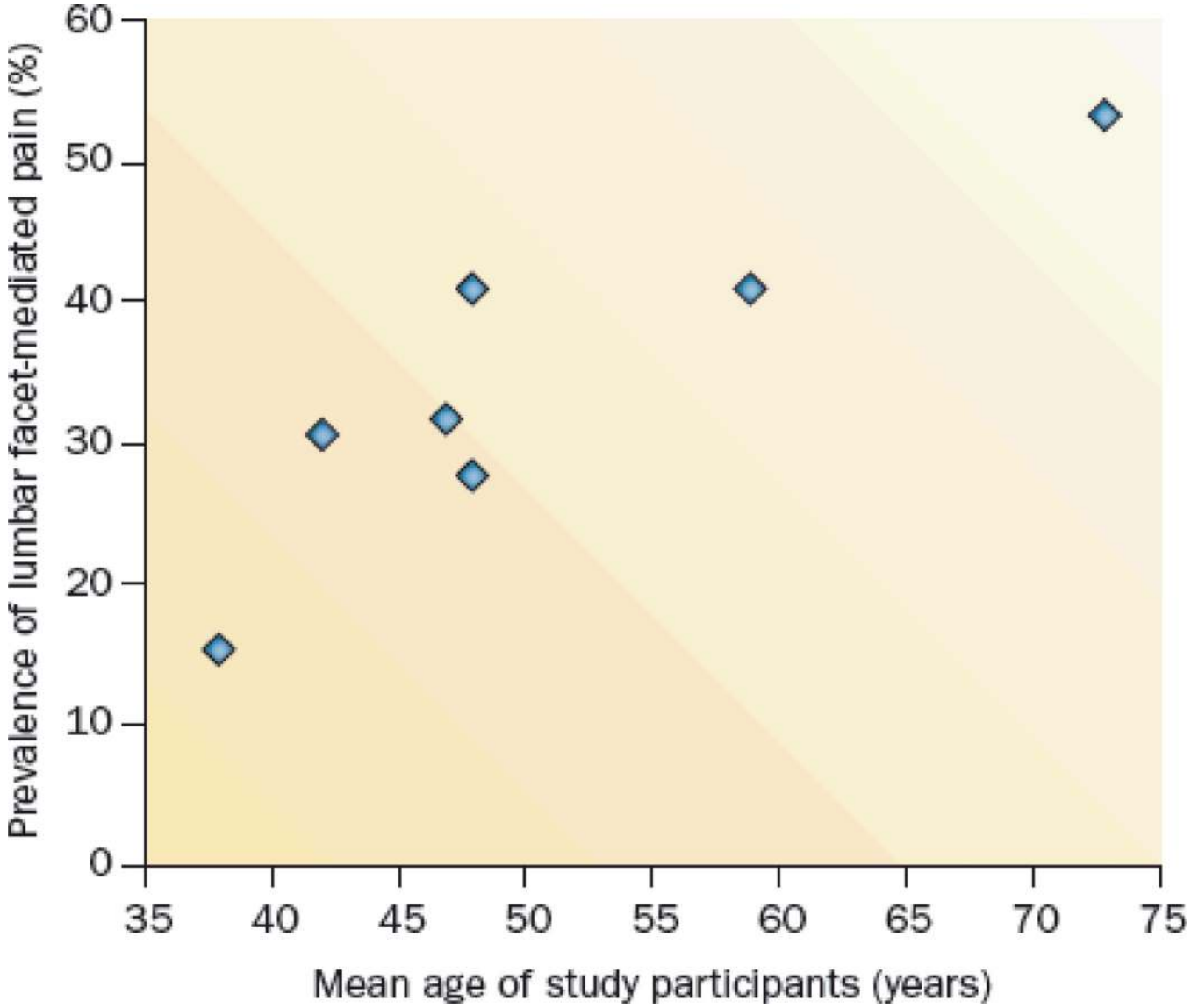

Figure 4.

Relationship between age and prevalence of low back pain attributed to lumbar facet joints. Prevalence of lumbar facet-mediated pain as determined by double-blind, comparative block techniques in different clinical populations.[17,76,84,88,90–92] A higher prevalence of facet-mediated pain is seen in samples of older adults as compared to younger adults. Each point represents a group rather an individual; ecological correlations could underestimate the variability seen on an individual level.

Table 1

Commonly used imaging grading systems for facet joint osteoarthritis*

Grading system by anatomical location	Imaging modality	Imaging features included							
		Joint-space narrowing	Osteophytosis of articular processes	Hypertrophy of acticular processes	Facet or joint irregularity	Sclerosis	Subchondral erosions	Subchondral cysts	Joint-space vacuum phenomenon
Cervical									
Kellgren-Lawrence[16]	Radiography	x	√	x	√	√	x	x	x
Lumbar									
Pathria[12]	Radiography, CT	√	√	√	x	√	x	x	x
Kellgren-Lawrence[16]	Radiography	x	√	x	√	√	x	x	x
Weishaupt[14]	CT, MRI	√	√	√	x	x	√	√	x
Framingham[15]	CT	√	√	√	x	√	√	√	√

* All scales are four-grade ordinal scales that apply different combinations of the imaging features specified in the table. No scales for thoracic facet joint osteoarthritis are commonly used.