**Lumbar Facet Syndrome**

“Lumbar facet syndrome” describes acute or chronic inflammation of a lumbar zygapophyseal joint. The facet joint is thought to be the source of pain in 15-45% of patients with chronic low back pain. (1-6) The literature is mottled with various explanations for the genesis of facet syndrome, including: capsular sprain, joint hypomobility, synovial cysts and degeneration. (7-9) The term “facet syndrome” shares many common characteristics with intersegmental joint dysfunction, to the point that some clinicians characterize facet syndrome as the medical equivalent of intersegmental joint dysfunction.

The lumbar facet joints are motion-restricting joints. The sagittal orientation of the upper lumbar facet joints help to limit rotation, while the more frontal orientation of the lower lumbar facet joints resist forward displacement. (10) The facet joints and adjacent soft tissues are richly innervated by the medial branches of the dorsal rami. (7,11-15) The facet capsule contains low-threshold mechanoreceptors and nociceptors that fire when the facet is compressed or its capsule is stretched. (17,18) Nociceptors can be hypersensitized by an inflammatory process. (19)

Repetitive capsular stress or other cumulative low-level trauma is the most common origin of facetogenic pain. (20) Excessive compression and extension of the lumbar spine (especially in the presence of degeneration) cause the “inferior articular process to pivot about the pars and stretch the joint capsule.” (7) Acute or repetitive trauma leads to inflammation and joint dysfunction, including intra-articular adhesions with subsequent degenerative changes of the facet joint. (21) The biomechanical three-joint model suggests that dysfunction involving any portion of the segmental tripod will lead to a cycle of self-perpetuating changes, including degeneration, in the remaining components.

Nociceptive stimulation of the facet joint has been shown to cause back and/or leg pain. (22-25) Not surprisingly, the most frequent complaint associated with facet syndrome is lower back pain radiating toward the flank, hip, and thigh. (20) While scleratogenous referral to the thigh is likely, true radicular complaints suggest an alternate pathology. (26,27) Since the medial branch of the dorsal ramus does not cross the mid line, facet pain generally presents in an ipsilateral fashion with peak intensity over the affected joint. (28-31) A complaint of stiffness or morning stiffness may be associated with degenerative change of the facet joint. (32-35) Symptoms of facetogenic pain may present following an acute injury but are more commonly the result of cumulative trauma.
The “1988 Volvo award in clinical sciences” study identified seven factors associated with the
diagnosis of facet joint pain: an older patient (over 65), with recurrent low back pain, not extending beyond the knee, which is maximally exacerbated with extension from a fully flexed position. Clinical findings demonstrate normal gait, with the absence of muscle spasm, and a negative Valsalva. (36) Other researchers have identified “relief with recumbancy” as another criteria for the diagnosis of facetogenic pain. (37-38) Predisposing factors for the development of facet syndrome include a history of trauma, overuse, osteoarthritis, systemic arthropathy, and being overweight. (39)

Clinical evaluation should demonstrate localized tenderness to palpation of the facet joint. (32,40,41) Muscle guarding may emerge as a protective response. (42) Range of motion testing will likely elicit pain in extension, as this causes compressive loading of the facets. (28,32,36,44) Extension combined with lateral flexion or rotation generates maximal compression of the facet joint and is even more likely to reproduce symptoms. (32,36,45) Facet syndrome is often accompanied by postural imbalances, including hyperlordosis and/or lower crossed syndrome.

While conditions like disc lesions and sacroiliac joint dysfunction have reliable orthopedic diagnostic tests, the diagnosis of lumbar facet syndrome lacks orthopedic evaluation with high sensitivity or specificity. (46) Application of a manual “springing” pressure over the affected zygapophyseal joint (Spring test) will likely provoke symptoms. (43) Spinal percussion and the Segmental rotation test may be useful in the diagnosis of facet syndrome. (43) Orthopedic evaluation of benign lower back pain most frequently demonstrates positive Kemp’s and Yeoman tests. (47) Neurologic testing is characteristically normal. Confirmatory medical diagnostic procedures include facet joint injection and fluoroscopically-guided nerve blocks. (32, 48-58) Diagnostic nerve blocks (with pain relief post-injection) are thought to be the most reliable diagnostic tool for lumbar facet pain. (58,59)

Evidence-based recommendations from the American College of Physicians (ACP) and the American Pain Society (APS) suggest that routine spinal imaging of benign lower back pain may be unnecessary. There are no radiographic findings that can identify the lumbar facet joints as the source of the patient’s symptoms. (59) Furthermore, radiographic findings do not correlate with facet-generated symptoms. (32,60- 63) Guidelines suggest “imaging only for patients who have severe or progressive neurologic deficits or signs or symptoms that suggest a serious or specific underlying condition” (64). Radiographs are appropriate for patients with “red flags” including a history of: cancer, unexplained recent weight loss, bone disease, systemic disease, inflammatory arthropathy (particularly ankylosing spondylitis), steroid use, immune suppression, fever, nocturnal pain, prior lumbar surgery, in patients with suspected congenital deficit or instability, and in those whose pain is usually severe, progressive, prolonged or unaffected by position. Radiographs may be needed to rule out suspicion of vertebral compression fracture or spondylolysis/ spondylolisthesis.
Likewise, advanced imaging is unable to predict lumbar facet joint pain. Advanced imaging, including MRI, should be reserved for patients with radicular complaints who are potential candidates for surgery or injections and only when the results of the test are likely to affect clinic decision making. Advanced imaging would be appropriate for patients with a history of major trauma, severe neurologic compromise, or suspicion of vertebral infection. CT scanning has no value in the diagnosis of lumbar facet pain.

Differential diagnostic considerations for lumbar facet syndrome include: intersegmental joint dysfunction, myofascial pain, spondylolysis, spondylolisthesis, sprain/strain, disc lesion, fracture, compression fracture, DJD/DDD, stenosis, neoplasm, infection, inflammatory arthropathy, sacroiliac joint dysfunction, hip pathology/osteoarthritis, abdominal aortic aneurysm and referred pain, particularly from the gastrointestinal or genitourinary systems.

Treatment progresses from pain relief through mobility and eventually functional stabilization. There is significant data to suggest that spinal manipulation (SMT) is an effective treatment for LBP. Spinal manipulation produces zygapophyseal joint gapping with subsequent break up of intra-articular adhesions. Elimination of zygapophyseal joint adhesions help to reestablish “normal” function and may slow the degenerative process. Spinal manipulation does not stress the facet capsule beyond its physiologic range and is thought to be safe. The only dose-response efficacy study of SMT for LBP suggested that 12 visits over 6 weeks provided the most favorable outcomes. Research has demonstrated that SMT is superior to alternate options including: traditional medical management (muscle relaxants, pain meds, anti-inflammatories), physical therapy, pain management, exercise, acupuncture, bed rest and massage.

Fritz identified five criteria that predict the success of spinal manipulation for lower back pain. These include pain lasting less than 16 days, no symptoms distal to the knee, low fear avoidance beliefs (FABQ score of less than 19), hip internal rotation greater than 35 degrees, and hypomobility of at least one lumbar segment. Related research suggests the first two factors are most significant.

SMT should address restrictions in the thoracic, lumbar, sacroiliac, and pelvic regions. Clinicians should be alert to the possibility of structural or functional instability, which may present in a very similar fashion to facet syndrome. Patients with micro- or macroinstability will benefit from spinal stabilization rather than manipulation, although facet syndrome and global instability are not always mutually exclusive.

Myofascial release techniques or IASTM may be appropriate for lesions in the lumbar erectors, quadratus lumborum, hip flexors, hip rotators, gluteal muscles, piriformis, and hamstrings as well as the iliolumbar ligament. Flexibility exercises may include a knee to chest stretch and hamstring stretch. Rehab of facet syndrome should focus on helping the patient find and maintain a neutral spine posture. The addition of spinal stabilization exercises may help to reduce pain, disability, medication intake as well as future episodes of lower back pain. Stability
exercises may include: side bridge, bird dog, dead bug and hip abductor strengthening (99). Postural correction may be necessary for lower crossed syndrome, and breathing exercises are appropriate for those with dysfunctional respiration.

Heat, ice, ultrasound, and e-stim may help relax muscles and provide short-term palliative relief in the early phases of facet syndrome treatment. (100) Patients may need to limit heavy physical activity but should avoid bed rest. (101) Lifestyle modifications should include removal of the activity that induces pain. Patients should be counseled on lifting mechanics, work activities, sleep positions and shoe wear. Educational counseling regarding predictable exacerbating activities for lumbar, lumbosacral, and hip trigger points is warranted. This may include minimization of prolonged sitting and sedentary hobbies. Yoga has been shown to be an effective treatment for simple mechanical low back pain (102). The addition of NSAIDS may help reduce pain and inflammation initially.

Medical management of facet syndrome includes fluoroscopically-guided intra-articular zygapophyseal joint steroid injections and radiofrequency ablation of the medial branch to (temporarily) eliminate all sensory input from the facet joint. (103) van Kleef (20) states “currently, the gold standard for (allopathically) treating facetogenic pain is radio frequency treatment. The evidence supporting intra-articular corticosteroids is limited: hence, this should be reserved for those individuals who do not respond to radio frequency treatment.”

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