European Journal of Radiology 84 (2015) 765–770

Contents lists available at ScienceDirect

European Journal of Radiology

journal homepage: www.elsevier.com/locate/ejrad

Clinical evaluation and surgical decision making for patients with lumbar discogenic pain and facet syndrome

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ARTICLE INFO

Article history:
Received 16 February 2014
Accepted 11 March 2014

Keywords:
Low back pain
Discogenic pain
Facet joint
Fusion

ABSTRACT

In industrialized countries, more than two thirds of the population suffers from low back pain (LBP) in their lifetime. LBP associated with lumbar disc herniation, stenosis, and instability is a well-known and documented entity. On the other hand, the lumbar discogenic pain and facet syndrome are difficult to be clearly identified, and they are not always detectable by imaging. This article describes the causes of these painful syndromes, which are typically without radicular component, explains the modern diagnostic procedures, and provides guidelines for surgical decision making.

1. Introduction

Low back pain (LBP) has a lifetime prevalence ranging from 11% to 84% in industrialized countries [1,2]. In most cases, the natural history is favorable and the majority of those affected do not require any medical help because pain regresses spontaneously within a short time [3]. LBP is defined "chronic" if it is persisting for at least 3 months [4]. The transition from acute to chronic pain is a poorly explained phenomenon [5]. One year after an acute episode of LBP, a fifth of patients complain of persistent pain, disabling in professional and private life [6].

Wieser et al. showed that in Switzerland lumbago was one of the main problems of health [7]. The prevalence of LBP was 50% on 2507 participants along more than 20 years. Medical costs amounted to a total of 2.6 billion euros (i.e. approximately 1842 euros/patient), which corresponds to 6.1% of gross domestic product. These costs were attributable to analgesics (28.1%), consulting to the family doctor (11.6%) and to specialists (6.4%), physiotherapy (7.6%), other manual therapies or alternative medicine (9.3%), as well as hospitalizations (6%) and surgical treatments (4.2%). Indirect costs, primarily related to loss of productivity and the ability to work, were even higher, of the order of 4.6 billion euros [7].

LBP may be related to several pathological entities, such as lumbar disc herniation, spinal stenosis and instability. Discogenic pain and facet syndrome, respectively, represent two particular subtypes of LBP. These two clinical conditions tend to occur separately in the lumbar spine. Indeed, only 3% of 92 patients had concordant pain on discography and pain relief after facet joint injection [8].

Patients with this kind of pain require comprehensive care by an interdisciplinary team of surgeons, rheumatologists, neurologists, neuroradiologists, physiotherapists, and specialists in pain management. In this paper, authors provide a review on pathogenesis, clinical findings, and surgical management of these syndromes. Furthermore, an algorithm concerning the surgical decision making is suggested.

2. Origin of pain

2.1. Discogenic pain

Discogenic pain typically origins from the intervertebral disc. The only innervated parts of the intervertebral disc are located in the outer third of the annulus fibrosus and in the adjacent vertebras' endplates [9]. The nucleus pulposus does not contain any nerve fibers. However, nociceptive fibers may come in contact with the nucleus pulposus when it is significantly degenerated and the annulus fissured [10]. In case of prolapse of the nucleus pulposus through the internal layers of the annulus (the so-called IDD: internal disc disruption), proteoglycans are released, and inflammation

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http://dx.doi.org/10.1016/j.ejrad.2014.03.016
0720-048X/

Table 1
Red flags for low back pain.

Red Flag	Presumptive diagnosis
1. >50 years or <20 old	Cancer, infection
2. History of tumor	
3. Unexpected loss of weight	
4. Immunosuppression	
5. I.V. drug abuse	
6. Fever, shivers	
7. Inflammatory back pain	
1. History of trauma	Vertebral fracture
2. Chronic steroids intake	
3. >70 years	
1. Urinary incontinence	Cauda equina
2. Fecal incontinence	syndrome
3. Saddle block anesthesia	
4. Progressive inferior limbs weakness	

Table 2
Waddell's signs.

Category	Signs
Tenderness tests	Nonorganic tenderness may be either superficial or nonanatomic: • Skin is tender to light pinch over a wide area • Deep tenderness is felt over a wide area
Simulation tests	Based on movements which give the patient the impression that a particular examination is being carried: • Axial loading: downwards pressure on the head • Rotation: rotate patient holding shoulders and hips in same plan
Distraction tests	Positive tests are rechecked when the patient's attention is distracted: • Straight leg raise causes pain when formally tested, but straightening the leg while doing Babinski does not
Regional disturbances	Weakness: multiple muscles not respecting the same root Sensation: loss of sensation does not respect a dermatome
Overreaction	Subjective reaction to the test depends on the emotion.

The presence of multiple Waddell's signs may suggest a behavioral component to a patient's pain.

cascade and pain follow, which are also induced by interleukin secretion. About 39% of all patients with chronic LBP have such IDDs, which can be identified by discograms [11].

2.2. Facet syndrome

In 1933 Ghormley, who first performed oblique spine radiographs to visualize the zygapophysial or facet joint, coined the term "facet syndrome", to refer to LBP with "sciatica" originating from the facet joints [12]. This radiation to the lower extremities seems to reflect the phenomenon of referred sclerotomal pain. The facet joints had been previously recognized as a potential source of pain [13], from when a referred pain may originate [14]. The facet joints have dual innervations: firstly, by the medial branch of the dorsal ramus of the spinal nerve [15,16] of the same level and secondly, through the dorsal branch of the segment above [17,18]. These branches innervate the multifidus muscle, the interspinous ligament, and the periosteum of the vertebral arch [15–17,19]. From a pathological point of view, the cause of facet syndrome is unknown. The facet joint may be affected by systemic disease, as rheumatoid arthritis and ankylosing spondylitis, or be site of microtraumatic fractures, osteoarthritis, meniscoid entrapment, synovial impingement, joint subluxation, synovial inflammation, loss of cartilage, and mechanical injury [20–22].

3. Clinical evaluation

First of all, when approaching a patient with LBP, the physician should be able to identify so-called "red flags". These signs or symptoms may be indicative for cancer, infection or trauma involving the lower spine (Table 1). Furthermore, we must keep in mind that nonorganic signs or "Waddell's signs" (Table 2) in patient with chronic LBP as psychological distress may cause an overreaction during the physical exam which does not corroborate anatomical and radiological signs [23].

3.1. Discogenic pain

Approximately 45% of low back pain appears to be of discogenic in origin [8]. Discogenic pain is triggered by activities leading to an increased pressure within the disc space. These activities include standing or sitting for prolonged periods, leaning forward, sneezing and coughing. Provocation of pain when rising from sitting seems to be correlated with discogenic pain as well [24]. Concerning the location of the pain, patients with painful discs usually complain of midline pain [24,25]. Some authors determined significant relationships between discogenic pain and centralization of pain (pain at the midline) during repeated movement testing, considering that centralization correlates strongly with a positive discography [24,26]. The centralization phenomenon refers to "the migration of low back and/or radiating pain to the spinal midline in response to specific position or movements" [26]. Other authors demonstrated in a prospective study that this centralization phenomenon has a high sensitivity but a poor specificity in patients with discogenic pain in the presence of a competent annulus [24,27]. On the other hand, peripheralization of the pain (pain in lower extremities) may indicate discogenic pain with a breached annulus [26,27]. An axial computed tomography post disc injection with saline solution permits to define the nuclear morphology and to determine the state of the nuclear envelope. Donelson et al., demonstrated in their prospective study that centralization correlates with an intact annulus, and peripheralization with an annulus not functionally competent because of radial and/or concentric fissures involving the outer third of the annulus [27]. However, none of the patients with facet joint pain reported centralization of pain in the prospective study of Young et al. [24].

3.2. Facet syndrome

In the literature, the lifetime prevalence of facet pain varies widely, ranging from 5% to 90% [28,29] in a population with LBP. This difference is due to the various available diagnostic methods and poor selection criteria. Using more rigorous dual blocks criteria (see below) the prevalence drops to 15% [30]. In an older population of patients referred from rheumatologists, the prevalence was 40% [31].

The clinical features of lumbar facet syndrome remain problematic to be identified. Pain onset at dorsal extension and release at flexion is often considered suggestive for facet pain, even if non-specific, such as maximal tenderness upon deep palpation of posterior elements. Effectively, there is no syndrome that discriminates between LBP caused by facet joint and that caused by other structures [32], because most physical examinations simultaneously test several structures including muscles, ligaments, discs and facet joints. Withal, Reven et al., proposed the followed seven variables for facet syndrome: age greater than 65 years and pain that was not exacerbated by coughing, not worsened by hyperextension, not worsened by forward flexion, not worsened when rising from flexion, not worsened by extension-rotation, and well-relieved by recumbence. The presence of five out of these seven variables allows selecting LBP that might be correlated with facet syndrome and relieved by facet joint block [32]. However, they