DISC DISEASE

A SUMMARY AND REVIEW

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Abstract: A review of the etiology, clinical, radiological and laboratory presentation, differential diagnosis and management goals of disc disease is presented.

Index Terms: Disc disease, disc herniation, disc bulge, disc prolapse, disc lesion, internal disc disruption, annular tear, chemical radiculitis, low back pain, sciatica, chiropractic.

SUMMARY:

The disc is the connective tissue between two vertebral end plates of hyaline cartilage. It consists of two different parts, the **annulus fibrosis and nucleus pulposus** (2).

The intervertebral disc has a number of functions (1). First, it separates the vertebral bodies. Secondly, it carries the weight transferred from the vertebra above. Thirdly, it is deformable to accommodate the rocking movements of the vertebrae and fourthly, it performs its normal movements without injury.

There is no clear boundary between the nucleus and the annulus within the disc. Rather, both parts merge (1). The annulus is made up of approximately 90 concentric layers of helicoid laminated fibres, with adjacent bands oriented in opposing directions. The nucleus is a gelatinous structure, composed mostly of a mucopolysaccharide gel (90% water) with small amounts of cartilaginous fibrils (6). The discs in general are primarily composed of collagen, proteoglycans and water. Collagen acts as the connective substance between disc and vertebrae as well as the basic building block of the annulus. The proteoglycans imbibe fluid, swell the nucleus and maintain resistance to load. The disc is generally avascular with nutrients supplied passively by diffusion via the endplates (6).

Disc disease may develop in the presence of bacterial infection, inflammations, neoplastic diseases, malacic states, systemic disease and particularly trauma. However, any cause or combination of causes which tends to alter vertebral structure will likewise modify and interfere with disc physiology (3).

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Disc pain is difficult to identify precisely because the origin or source of pain is so deep (12). Indeed with spinal pain in general corroboration between clinical observations and histologic studies is generally impossible and there is often no modification of the painful tissue identifiable through current methods of imaging (4). This statement particularly holds true for the disc. Although in recent times more light has been shed on internal disc disruption and its imaging by CT/Discography (5).

In summary, the disc can be a source of pain, internally through annular disruption or externally by herniating.

A. ANNULAR DISRUPTION:

The works of Yoshizawa (7) and Bogduk (8) have established that the outer one-third of the annulus has a nerve supply. This then provides an anatomical basis for pain emanating from this region of the disc. A view borne out by the reproduction of pain by provocation discography (5).

Degeneration of the disc is thought to be a common cause of low back pain, and discs are now thought to degenerate before facet joints (66), with increasing age a related factor (2).

However, apart from normal aging the discs are subject to two major forms of mechanical injury:

Torsion injury with potential tears of the annulus (9)(15), and compression injury with the formation of Schmorls nodes and their attendant sequelae (9)(10).
In torsion injury Bogduk and Twomey (9) postulate that excessive torsion stresses the annulus and both zygapophyseal joints with a spectrum of resulting lesions. With respect to the disc they suggest that the resulting lesions are circumferential splits between the outer lamellae of the annulus. Repeated injuries forming radial fissures. Farfan states that torsion causes discal distortion and eventual avulsion of the annulus from the end plate (18).

It is recognised that the disc can withstand extensive compressive force without herniation (11), however Schmorl's nodes may result from such force (10), forcing disc material into the vertebral end plate. Bogduk (12) again postulates that this type of injury sets off a chain of events which may lead to herniation of the disc. He suggests that when nuclear material enters the vertebral body, the protein constituents of the nuclear matrix may elicit and antigenic response. This sets off an insidious inflammatory action back into the nuclear material itself with degradation of proteoglycans. The process spreading centrifugally to affect the annulus, causing "internal disc disruption" a term coined by Crock (13).

Indeed, Crock also supports the theory of these types of disc lesions that penetrate into the vertebral body and an autoimmune reaction (14).

If nuclear material leaks from a diseased disc and contacts nerve roots and other perineural tissue a "chemical radiculitis" may result (16)(17).

The clinical features of internal disc disruption are poorly localised low back pain, possibly lower limb pain, pain and stiffness on movements which stress the annulus (eg. sitting), even loss of energy, weight loss and depression (12)(14).

B. NUCLEAR HERNIATION:

If a large enough defect occurs in the annulus a disc bulge will occur. Should this defect worsen the annulus may not be capable of constraining the nuclear material and a herniation may result (19)(20)(24). The incidence of this even as a cause of back pain may be a slow as 1% (26). Jinkins in a study of 236 disc extrusions, found that 56.4% were posteriorly placed, 29.2% were anteriorly placed (ie anterior to the neural foramen), and 14.4% were centrally located (ie. into the vertebral bodies) (21).

Not all disc herniations are symptomatic (22). When they are, the pattern of pain is dependant on the anatomy that the herniation involves both mechanically and chemically. Apart from direct nerve root pressure the anatomic basis of disc extrusion pain rests partially with somatic fibres originating from the recurrent meningeal nerve which supplies the posterior longitudinal ligament, the meninges, blood vessels, a portion of the vertebral body periosteum and underlying bone (21)(23). It also involves afferent fibres of the anterior and anterolateral disc and paradiscal structures which project immediately to the paraspinal sympathetic ganglia, giving not only pain but potentially aberrant vasomotor, pilomotor and sudomotor activity (21).

Classically, the most common symptoms associated with nuclear herniation are radicular pain in particular sciatica (25). This may be accompanied by spasm and stiffness in the low back and parasthesiae, numbness, weakness and/or atrophy in the lower extremities (27). In addition Jinkins et al (21) further suggest that disc extrusion may be responsible for an "autonomic syndrome" with symptoms including diaphoresis, piloerection, vasomotor changes, changes in blood pressure, heart rate, respiratory rate and alertness.

C. SCIATIC RADIATION:

Pain to the lower extremities may arise from nearly every anatomical structure in the lumbar spine (23). True sciatica (radicular pain) should be ascribed to root compression only with its attendant symptoms of defined leg pain, numbness, weakness or paraesthesia (28)(28A).

Where there is no neurological deficit and the pain is felt in a sciatic distribution the term sciatic distribution should be used. Internal disc disruption when severe can refer pain into the legs (12).

It should be noted however, that in patients with sciatic radiation, radiculopathy cannot be ruled out solely on the basis of a clinical examination. Haldemann et al found that the most useful test for determining the presence or absence of radiculopathy was electrodiagnostic testing (29).

DIFFERENTIAL DIAGNOSIS:

A differential diagnosis of lumbar disc disease should include all other somatic causes of low back pain and leg pain especially the facet joints, myofascial pain and hip degeneration. Other conditions to be considered are spinal stenosis, tumours, infection, vascular disorders such as abdominal aortic aneurism, rheumatoid arthritis, ankylosing spondylitis, Pagets disease, herpes zoster prior to vesicular eruption, visceral referred pain and psychogenic pain. (30).

USUAL AND CUSTOMARY EXAMINATION PROCEDURES:

Physical/Orthopaedic Examination.

In true disc herniation the classical findings on physical examination are low back pain radiating to the lower extremity with neurological deficit in the affected leg (25)(25A). The pain is accentuated by straining and relieved by recumbency (25A). However, other classical findings such as are few and far between.

There is considerable overlap between the clinical presentation of low back pain of differing origins. Some tests that may assist in indicating a "non-contained" disc lesion are nerve root traction tests such as low back pain on straight leg raising (Lasegue test) (31) and cross leg pain on straight leg raising (32). The Bowstring test where at maximum straight leg raising, the knee is flexed a few degrees and digital pressure is applied over the lateral popliteal and posterior tibial nerves at the knee posteriorly (28A).

Also pain on prone lying knee flexion which stretches the third lumbar nerve root (33).

There is usually pain on several lumbar movements which aggravate the herniation with the pain being felt in the low back, leg or both. However in chronic herniation lumbar ranges of motion may not elicit pain.

In contrast to the above a contained disc lesion (internal disc disruption) produces back pain of a non specific nature. When severe it could be referred to the lower limbs but without neurological signs. Pain is exacerbated by any movement which stresses the annulus. Muscle guarding could be a feature (12).

DIAGNOSTIC IMAGING:

The diagnostic imaging modalities used in the assessment of disc disease include plain film radiography, myelography, computerised tomography (CT), discography (with or without provocation), magnetic resonance imaging (MRI) and combinations of the above notably CT with myelography and CT with discography (Disco-CT)(34).

1. Plain film radiography will not show the disc itself but will demonstrate the late manifestations of disc disease which include: spondylosis, disc space narrowing and vacuum phenomenon. Certainly plain radiographs demonstrate many types of pathology which may give rise to back pain and indeed some authors advocate plain radiographs in every case of low back pain and sciatica (67).

2. Myelography is now primarily used to demonstrate small intra-thecal masses and adhesive arachnoiditis (35). However, it is still used by some to image posterior disc protrusion prior to surgery (36).

3. Computed Tomography provides excellent images of non-contained disc extrusion both annulus bulging and nuclear herniation. It can also image internal disc disease to the extent of visualising vacuum phenomenon (34)(36)(37).

4. Discography is a useful tool in the assessment of patients with disc degeneration (38)(39). However, it is invasive and not without attendant risks especially infection (40).

5. Magnetic Resonance Imaging can be used to measure quantitatively degeneration in discs (41) and is thought to depend on the change in the water content in the disc with aging (42).

6. CT/Myelography is often favoured rather than conventional myelography where CT findings are equivocal, especially in post-surgical circumstances where differentiation of recurrent disc herniation from scar tissue is important (34).

7. Discogram with CT. According to Jackson et al (43) disco-CT has a high rate of accuracy and is recommended in selected patients with suspected lumbar disc herniation whose other tests are non-diagnostic, especially those with possible foraminal or recurrent herniation. It is also considered the diagnostic imaging of choice for internal disc disruption (12)(44).

CLINICAL LABORATORY TESTS:

With disc herniation there is not specific blood test available. Marshall et al (16) suggested that a rupture of the annulus with consequent liberation of nuclear fluid into the tissues would be followed by a high serum titre of antibodies to glycoprotein at three week post rupture. However, the authors only isolated this phenomenon in one case and the test has not gained credence.

Electro-diagnostic testing can be used in the investigation of spinal nerve root function. It is possible to determine with reasonable accuracy the segmental level of a radiculopathy and to isolate both sensory and motor components that may exist independently or at the same time (45).

GOALS OF TREATMENT:

1. Reduction of pain, inflammation and spasm.
2. Restoration of function.
3. Reduce risk of reoccurence.

CHIROPRACTIC MANAGEMENT:

The selection of a management regime for active disc disease should be based on the category of pain presentation, duration and severity of symptoms and the amount of anatomic change.

The clinical presentation of disc disease can be summarised in the following way:

1. Acute low back pain.
2. Sub-acute low back pain.
3. Chronic low back pain.
4. Acute low back pain with referred pain into the buttock, hip or thigh.
5. Sub-acute low back pain with referred pain into buttock, hip or thigh.
6. Chronic low back pain with referred pain into the buttock, hip or thigh.
7. Acute low back and leg pain including the lower leg.
8. Sub-acute low back and leg pain including the lower leg.
9. Chronic lower leg pain with or without low back pain.

Where acute is defined as 1-10 days.
Where sub-acute is defined as 11 days-6 weeks.
Where chronic is defined as >6 weeks.

A Guide to Chiropractic Treatment of Each Category:

Categories 1,4,7:

Initially 2 days of bed rest is recommended (46), however up to 2 weeks may be required (47). treatment should be directed at relieving symptoms with physical therapy modalities such as TENS at home (48) and anti-inflammatory/analgesic medication when required (49). As the patient improves mobilising techniques and exercises may be implemented, initially to increase ranges of motion and enhance disc nutrition (50). However, where neurological changes such as with cauda equina syndrome exist surgery may be indicated immediately.

Categories 2,5,8.

If we assume that the regime above for the acute phase has been instituted and was not curative, then a trial of traction should be considered, using static traction at first then progressing to intermittent motorised traction (52)(53). Importantly in this sub-acute phase the patient should maintain aerobic fitness where possible and a tailored functional exercise programme instituted (54).

Categories 3,6,9.

The treatment of the chronic phase depends upon many factors including severity of dysfunction, psychological factors and neurological deficit.

Certainly, if symptoms are not resolving at the three month mark there is evidence that further delay of surgical intervention in true disc herniation with sciatica may inhibit functional restoration (55). However, Halkelius (55) showed that sciatica is a transient and self limited condition that usually resolves satisfactorily regardless of whether the treatment is conservative or surgical. Yet in the chronic sciatica type of patient with unremitting pain, surgery does seem to offer a better long term prognosis with respect to their pain, residual sciatica and frequency of reoccurrences (55).

Where the patient is progressing slowly, management should concentrate on functional restoration, especially strength, mobility and endurance work. Also on pain management techniques, ergonomic advice, return to suitable work and psychological support (50).

The management programme may involve the treating chiropractic physician, psychologist, occupational therapist and back school.

Progress in the chronic stage may be marked by exacerbations and remissions. Treatment for exacerbations depends on what level the patient regresses to.

PROGNOSIS:

The natural history of low back pain is benign and it is generally a self limiting condition. In fact 90% of all low back pain episodes resolve without physician intervention (56). The natural course for disc herniation is also favourable (57) with most resolving satisfactorily within 6 months (2).

DISC DISEASE AS A COMPLICATION:

Disc disease has a number of individual risk factors:

1. TRAUMA. Whether a single traumatic event or a repetitive strain, trauma has been found to predispose to disc disease (12).

2. PHYSICAL FITNESS. Improved physical fitness has a preventative effect on the occurrence of disc disease (58).

3. HEIGHT AND WEIGHT. Taller people and the obese are more prone to disc disease (2).

4. SMOKING. Smoking has been linked to disc disease (59)(60).

5. SPINAL DISEASES. Spinal disease such as Scheuermanns disease have been linked to disc disease (61).

6. HEREDITY. Lawrence claims a link between relatives of patients with disc herniation (62).

7. AGE. The risk of disc herniation at the L4/5 and L5/S1 levels increases until the fifth decade and thereafter decreases. However, the relative risk of disc herniations at L2/L3 and L3/L4 is greater in the population over the age of 50 years (62). Lysosomal enzymes have been identified in the disc and the balance between these and biosynthetic activities may be an important factor in determining tissue changes of the disc with age (65).

8. SEX. Most surveys indicate the relative risk of low back pain is similar for males and females until the age of sixty (64).

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