Lumbar Canal Stenosis: A Review Article

C Mauffrey, P Makrides, C Lewis, M Brewster

Citation

C Mauffrey, P Makrides, C Lewis, M Brewster. *Lumbar Canal Stenosis: A Review Article*. The Internet Journal of Spine Surgery. 2006 Volume 4 Number 1.

Abstract

Lumbar canal stenosis is the narrowing of the spinal canal or the tunnels through which nerves and other structures communicate with that canal. This can lead to claudication or radiculopathy. In this article, we review the literature for the various modalities of treatment.

INTRODUCTION

Lumbar canal stenosis is the narrowing of the spinal canal or the tunnels through which nerves and other structures communicate with that canal. This can lead to claudication or radiculopathy.

ANATOMY

The spinal column is made up of individual vertebrae, which articulate body to body and between the articular facets. The vertebral laminae are joined together by the ligamentum flavum. The spinous processes are joined by the supraspinous and interspinous ligaments. The articular facets are reinforced by the articular ligament. Adding further stability are the anterior and posterior longitudinal ligaments, which run along the whole length of the spine.

The vertebral bodies are joined by the intervertebral discs, which provide great stability to the spine. They comprise of the annulus fibrosus peripherally and the central nucleus pulposus.

The vertebral canal is surrounded by a vertebral body and a neural arch. The neural arch consists of the pedicle on either side. Each pair of pedicles supports a lamina, which forms the posterior wall of the central canal.

At the level of the intervertebral discs however, the annulus fibrosus forms the anterior border of the canal, the facet joints form the postero-lateral border and the ligamentum flavum forms the posterior border.

PATHOPHYSIOLOGY

Spinal stenosis was classiffied by Arnoldi in 1976 into congenital and acquired causes. Congenital causes may result from narrow lumbar canals due to short pedicles, thickened lamina and facets, or hyperscoliotic or lordotic curves. Most commonly, lumbar canal stenosis is due to acquired degenerative or inflammatory changes (arthritis) of the intervertebral discs, ligaments and facet joints. These changes include cartilaginous hypertrophy of the articulations surrounding the canal, intervertebral disc herniations or annular bulges, hypertrophy of the ligamentum flavum and bone spur (osteophyte) formation.Jane et al [1] have suggested that the minor instability (referred to as micro-instability) at the articular surfaces surrounding the canal cause the pathologic changes that result in lumbar canal stenosis. Micro -instability is defined as abnormal movement in the vertebral joints. Despite the fact that the movements are small and clinically silent they can lead to weakening of the joint capsule and consequent reactive changes such as osteocartilaginous hypertrophy, ligamentum flavum thickening or calcification, or subluxation of the vertebral joints. These can all result in lumbar canal stenosis. Canal stenosis can consequently lead to compression of the blood supply to the nerve roots. This can lead to neurogenic claudication. Two major types, were classified by Wilson: postural or ischemic [2]. Ischaemic neurogenic claudication results from failure to meet oxygen needs of the nerve roots due to a compressed canal during exercise. Postural claudication will result from positional accentuation of the stenosis. Other aetiological causes leading to spinal canal stenosis include Paget's disease, hyperparathyroidism, spinal tumours, infection such as TB, surgery and trauma.

CLINICAL HISTORY

The earliest complaint is non- specific back pain and leg

pain associated with numbness and weakness. The leg pain is commonly bilateral involving the buttocks and the thighs exacerbated by leg exercise. Other symptoms include burning and cramping of the lower extremities which may be mild and can progress to become disabling. Symptoms are typically worse when standing and patients have a characteristic gait as they stoop forward to improve their symptoms. This is characteristic of neurogenic claudication. Symptoms are releived by squatting position as this flexes the trunk and widens the lumbar canal [3] . Rarely other symptoms include visceral disturbances such as urinary incontinence and cauda equina syndrome. Finally there may be radicular signs from narrowing of the lateral recesses or the neural foramen in foraminal stenosis.

INVESTIGATIONS

Simple radiological investigations may reveal degenerative changes such as bone spurs, decreased disc space and facet hypertrophy in older patients. CT scan will show a more detailed picture of the bony anatomy. It is less accurate than an MRI in estimating the degree of compromise of the soft tissue elements unless combined with a myelogram. Thus the CT can underestimates the degree of stenosis. MRI is the preferred modality for evaluating and diagnosing lumbar stenosis. It allows visualization of soft tissues, including the neural elements, ligaments, epidural fat, subarachnoid space and intervertebral discs.

NATURAL HISTORY

Johnsson et al, studied the course of 32 untreated patients with spinal stenosis over 49 months. [4] They found that 15% improved, 70% remained unchanged and 15% deteriorated using the visual analogue scale. Using the same cohort of patients, based on clinical assessment, it was found that 41% improved, 41% remained unchanged and 18% worsened. Atlas et al. concluded that in patients with severe lumbar spinal stenosis surgical management was associated with higher patient satisfaction compared to non-surgical treatment. This multi centre prospective observational study evaluated patients over a 4-year period. Over time, there was convergence of the relative satisfaction from the treatment received [5] . Recent studies conclude that the natural progression of the disease is highly variable. 15% of patients were found to improve, 30% worsen and require surgery and 45% of the patients remain the same [6] .

TREATMENT

Management of lumbar spinal stenosis incorporates a wide spectrum of interventions. Depending on the severity of the symptoms, and the condition of the patient, treatment can be surgical or non surgical. Non surgical intervention includes bed rest, activity modification and physiotherapy. Medical treatment includes analgesia, nerve pain modifiers, epidural steroids and root blocks. Porter et al have reported that Calcitonin is effective in relieving symptoms of neurogenic claudication for some patients [7] . Alternative medicine such as acupuncture is also in use. Amudsen et al. failed to identify benefit from surgical or conservative treatment. They conclude however, that patients who were initially treated conservatively with an unsatisfactory result can still be treated surgically later with a good outcome [8] . Indications for surgical intervention include failed conservative treatment, intractable leg pain, progressive neurological deterioration and stenosis shown on imaging.The risk of surgical intervention will depend on the number of stenotic segments additional comorbidities, location of the stenosis. Furthermore scarring from previous surgery can render the operation very difficult. Risks are also higher in patients with iatrogenic instabiility and anatomical difficulties like kyphosis or scoliosis. Complications of laminectomy include wound infection, haematoma formation, cerebrospinal fluid leaks resulting form tears in the dura, meningitis, nerve root damage and the risk of postoperative spinal instability. Mortality from decompressive laminectomy is approximately 1 percent [9] .

Hansraj et. al. suggested that in simple spinal stenosis, which is degenerative stenosis (< grade 1 spondylolisthesis, <20 degrees scoliosis, no surgery) without radiological instability, decompressive surgery is most beneficial. He found that in complex spinal stenosis which is instability and post laminectomy (> grade 1 spondylolisthesis, >20 degrees scoliosis), junctional stenosis decompression and fusion are more efficacious [10] . Decompression laminectomy is performed through an incision in the midline at the stenotic levels. The operation involves removal of the hypertrophied ligamentum flavum and the posterior parts of the lumbar spinal canal, which are namely the pedicles, laminae and spinous processes. In cases where there is osteophyte formation, medial parts of the facet joints are removed. This can cause instability, especially in cases where facectomies have to be bilateral [11] . Abumi et. al. suggested that medial facetectomy did not affect the stability of the lumbar spine. On the contrary, total and unilateral facetectomy may cause lumbar spine instability [12] . Haher et al. further confirmed that destruction of the facet joint puts extra mechanical strain to the neighboring discs enhancing degeneration [13] . Lee et al, used a ligamentous, nonlinear, sliding contact, three-

dimensional finite element model to conclude that total laminectomy with facetectomy caused an increase in spinal instability. Unilateral laminectomy was found to have the best results with respect to stability [14] . A less invasive procedure with good post operative results is multi-level laminectomies. In this procedure the superior aspect of the inferior lamina and the inferior aspect of the superior lamina of the stenotic canal are removed, creating fenestrations, thus minimizing the risk of instability. This procedure is most suitable for patients with congenital stenosis, and mild to moderate degenerative stenosis [15] . Furthermore, expansive lumbar laminoplasty can be used in younger patients. This procedure involves the removal, followed by loose reattachment of the posterior vertebral arches. This procedure preserves stability and allows decompression and stabilization [16] . Matsui et. al. conducted a study looking at the post-operative results of expansive lumbar laminoplasty over 5.6 years, and found that 80% of the patients had excellent results [17] .

Distraction laminoplasty is a variant of laminectomy that allows maximal bone preservation. In this procedure distraction force is applied, in conjunction with an undercutting laminoplasty [18] . This procedure has not been extensively investigated and few clinical results have been reported.

Decompression through a Port Hole is essentially an open laminectomy, using a micro-dissection technique that spares the integrity of the posterior elements of the spinal canal. In a study by Kleeman et al. 54 patients were reviewed at four years showing that 96% had good results, with less back pain and a quicker recovery period [19] .

Another technique for the management of lumbar canal stenosis is spinous process osteotomy. This procedure involves the unilateral multifidus takedown, followed by osteotomy of the spinous process at the stenotic levels. This is then retracted en-bloc and “trumpeted” decompression is carried out. This technique is thought to offer good visualization and minimized tissue damage. It has been shown to decrease pain levels by 66% and improve outcome scores by 47% [20] .

Finally, spinous process distraction is another method described. This involves the use of a spinal implant placed between the spinous processes at the stenotic levels. This destructs the spinous processes and holds them in flexion. This is beneficial for high risk patients, as it can be performed under local anaesthesia [21] . A further study has shown that this method has significantly better results compared to conservative management over 1 year. Furthermore, it compares favourably with the success rates of decompressive laminectomy success rates, but has significantly less morbidity [22] .

DEGENERATIVE SPONDYLOLISTHESIS

In degenerative spondylolisthesis, the surgical options include decompression alone and decompression with fusion. Studies have shown that posterior decompression with fusion of the spinous processes at the stenotic level is the surgical option with the best outcome irrespective of age, compared to total facectomy and facet preservation [23] . Herkowitz concluded that the outcome is better if concomitant arthrodesis is performed [24] .

The use of instrumentation in lumbar canal stenosis:

The use of lumbar spinal fixators has a higher fusion rate [25] , and an improved functional outcome and alignment [26] . In his study, Mardjetko concluded that the satisfaction rate in patients who had undergone spinal fusion was greater (90%) compared to patients who had undergone decompression alone (69%) [27] . Clinical instability is uncommon following decompression but if more than 50% of the facet joint is excised, instrumented fusion in indicated [28] . Instrumented fusion has been recommended for recurrent stenosis as shown by multiple studies [29] .

The management of lumbar spine stenosis is extremely varied. Patients should be treated according to their co morbid and psychological factors. It is well established that outcome from various treatment are subjective and depend on patient's expectations. Furthermore studies are needed, and especially randomized control trials in order to assess and compare the various modalities of treatment of this common condition.

CORRESPONDENCE TO

Mr. Mauffrey, 115 Masons way, B927JF Solihull, England. Tel: +44 7738671101, Email: cmauffrey@yahoo.com.

References

1. Jane JA Sr, Jane JA Jr, Helm GA, Kallmes DF, Shaffrey CI, Chadduck JB, et al. Acquired lumbar spinal stenosis. In: Clinical neurosurgery. Baltimore: Williams & Wilkins, 1995:275-99
2. Wilson CB. Significance of the small lumbar spinal canal: cauda equina compression syndromes due to spondylosis. 3: Intermittent claudication. J Neurosurg 1969;31:499-506.
3. Alarez J., Hardy R. H., Lumbar spine stenosis: a common cause of back and leg pain. American Family Physician 1998
4. Johnsson KE, Rosen I, Uden A (1992) The natural course

of lumbar spinal stenosis. Clin Orthop 279: 82–86
5. Atlas, S. J, Keller, R. B., Robson, D., Deyo, R. A., Singer D.: Surgical and Nonsurgical Management of Lumbar Spinal Stenosis: Four-Year Outcomes From the Maine Lumbar Spine Study. Spine. 25(5):556-562, March 1, 2000
6. Swezey RL :Outcomes for lumbar stenosis: A five-year follow-up study. J Clin Rheumatol, 1996
7. Porter RW, Hibbert C: Calcitonin treatment for neurogenic claudication. Spine 1983 Sep;8(6):585-92
8. Amundsen, Tom, Weber, H., Nordal, H J., Magnaes, B., Abdelnoor M, Lilleas, F.: Lumbar Spinal Stenosis: Conservative or Surgical Management? : A Prospective 10-Year Study:Spine. 25(11):1424-1436, June 1, 2000.
9. Tuite GF, Stern JD, Doran SE, Papadopoulos SM, McGillicuddy JE, Oyedijo DI, et al. Outcome after laminectomy for lumbar spinal stenosis. Part I: clinical correlations. J Neurosurg 1994;81:699-706.
10. Hansraj KK, O'Leary PF, Cammisa FP Jr, Hall JC, Fras CI, Cohen MS, Dorey FJ:Decompression, fusion, and instrumentation surgery for complex lumbar spinal stenosis. Clin Orthop Relat. Res.2001 Mar;(384):18-25.
11. Boden SD, Martin C, Rudolph R et. al.: Increase of motion between lumbar vertebrae after excision of the capsule and cartilage of the facets. A cadaver study.The Journal of Bone and Joint Surgery, Vol 76, Issue 12 1847-1853
12. Abumi K, Panjabi MM, Kramer KM, Duranceau J, Oxland T, Crisco JJ.:Biomechanical evaluation of lumbar spinal stability after graded facetectomies. Spine 1990 Nov;15(11):1142-7.
13. Haher TR, O'Brien M, Dryer JW, Nucci R, Zipnick R, Leone DJ.:The role of the lumbar facet joints in spinal stability. Identification of alternative paths of loading. Spine 1994 Dec 1;19(23):2667-70
14. Lee KK, Teo EC: Effects of laminectomy and facetectomy on the stability of the lumbar motion segment. Med Eng. Phys. 2004 Apr;26(3):183-92
15. Postacchini F, Cinotti G, Perugia D et. al.: The surgical treatment of central lumbar stenosis. Multiple laminotomy compared with total laminectomy. Journal of Bone and Joint Surgery - British Volume, Vol 75-B, Issue 3, 386-392
16. Tsuji H., Itoh T., Sekido H.: Expansive laminoplasty for lumbar spinal stenosis. International Orthpaedics, 1990, Vol.14 (3): 309-314
17. Matsui H., Kanamori M., Ishihara H.,et. al., Expansive lumbar laminoplasty for degenerative spinal stenosis in patients below 70 years of age. European Spine Journal 1997. 6(3):191-196.
18. O'Leary P., McCance S.: Distraction Laminoplasty for Decompression of Lumbar Spinal Stenosis. Clinical Orthopaedics & Related Research 2001. 384:26-34.
19. Kleeman T., Hiscoe A., Berg E.: Patient Outcomes After Minimally Destabilizing Lumbar Stenosis Decompression: The “Port-Hole” Technique. Spine, 2000. 25(7):865-870.
20. Weiner B, Fraser R., Peterson M.: Spinous Process Osteotomies to Facilitate Lumbar Decompressive Surgery. Spine 1999, 24(1):62-66
21. Zucherman F.,Hsu K.,Hartjen C., et. al.: Treatment of lumbar spinal stenosis with an interspinous spacer. The Spine Journal 2002, 2(5):52-53.
22. Zucherman F.,Hsu K.,Hartjen C., et. al.: A prospective randomized multi-center study for the treatment of lumbar spinal stenosis with the X STOP interspinous implant: 1-year results. European Spine Journal 2004, 13(1):22-31.
23. Lombardi JS, Wiltse LL, Reynolds J et. al.: Treatment of degenerative spondylolisthesis. Spine 1985 Nov;10(9):821-7.
24. Herkowitz HN, Kurz LT: Degenerative lumbar spondylolisthesis with spinal stenosis. A prospective study comparing decompression with decompression and intertransverse process arthrodesis. The Journal of Bone and Joint Surgery, 1991, Vol 73, Issue 6 802-808
25. Yuan HA, Garfin SR, Dickman CAet al. A Historical Cohort Study of Pedicle Screw Fixation in Thoracic, Lumbar, and Sacral Spinal Fusions. Spine. 1994;19(20 Suppl):2279S-2296S.
26. Yone K., Sakou T., Kawauchi Y., et. al.: Indication of Fusion for Lumbar Spinal Stenosis in Elderly Patients and Its Significance. Spine, 1996, 21(2):242-248.
27. Mardjetko SM, Connolly PJ, Shott S: Degenerative lumbar spondylolisthesis. A meta-analysis of literature 1970-1993. Spine 1994. 19(20 Suppl):2256S-2265S
28. Hazlett JW, Kinnard P.: Lumbar apophyseal process excision and spinal instability. Spine 1982;7(2):171-6.
29. Herno A.: Surgical results of lumbar spinal stenosis. Ann Chir Gynaecol. Suppl 1995;210:1-969

Author Information

C. Mauffrey
Specialist Registrar Trauma and Orthopedics, University Hospital Coventry and Warwick

P. Makrides
Senior House Officer Orthopedics, University Hospital Coventry and Warwick

C. Lewis
Specialist Registrar Trauma and Orthopedics, University Hospital Coventry and Warwick

M. Brewster
Specialist Registrar Trauma and Orthopedics, University Hospital Coventry and Warwick