Investigating non-operative treatment options for lumbar spinal stenosis

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Lumbar spinal stenosis (LSS) is the most common reason for spinal surgery in the aging population accounting for approximately a \$1 billion/year in inpatient expenses [3, 6]. The socioeconomic impact is major concern in the Western societies. This is also a group of patients with significant comorbid conditions, which eliminate the surgical option, however nonsurgical treatment alternatives are few and poorly studied. In the upcoming 2006 issue of European spine journal, Tafazal et al. [12] in their article titled, “randomised placebo-controlled trial on the effectiveness of nasal salmon calcitonin in the treatment of lumbar spinal stenosis,” offer yet another interesting assessment on calcitonin use as the treatment option for LSS. Unfortunately, the study confirms our earlier work that in the nasal form, calcitonin is ineffective, at least in the short-term. Furthermore, this is one of the significant steps in exploring and assessing the non-operative alternative of calcitonin and its value in treating LSS, which the authors have pertinently described in the current paper. Our comments here are based on the premise of an identical study that was carried out by us with similar design and methodology published in 2004 [8].

The authors describe the results in 40 patients diagnosed with LSS and evaluate the placebo effect in comparison to active salmon calcitonin nasal spray in the treatment of lumbar spinal stenosis. The results

indicate that after the first 4 weeks during which patients received active versus placebo nasal calcitonin, there was no statistically significant difference in the change in outcome scores between the two groups.

In our opinion there is scope for wider research that is of potential importance to describe the measurement properties of calcitonin in spinal stenosis. Our study evaluated the efficacy primarily at 6 weeks. The open-label phase of the study in the second 6 weeks suggested a trend towards improvement in patients treated with salmon calcitonin during the second phase of the trial, particularly in pain scores and SF-36. It is conceivable that the beneficial effects of nasal calcitonin require a longer period than 6 weeks to become demonstrable, and efficacy may be achieved using a different treatment schedule. The authors should have explored this particular timeline effect. We agree that a greater volume of studies provide more data that can be used to shape clinical decisions. This increased amount of research also increases the chance that the results of some studies, at times, may conflict with results of other studies. As the number of studies on a particular issue grows, the potential for conflicting results increases. However, the current study by Tafazal et al. illustrates similar results to our study as acknowledged in the paper. Authors wishing to reach the widest possible audience, or a variety of specific audiences, may seek to report a single definable body of research in more than one paper, in repeated reports of the same work, in fractional reports, or in reports in more than one language [4]. However, this has less noble implications and close similarity to design and methodology, should hence be avoided [5]. Nonetheless, there have been limitations in both studies, which need further investigations; inadequate numbers of

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patients, nasal and not subcutaneous (SQ) drug testing compared to previous studies, and short-term follow-up.

The authors do not provide explanation as to why at the end of the trial, the VAS score for ‘leg pain’ actually deteriorated by 15 mm in the calcitonin group and 11 mm in the placebo group. Also, unexplained is the report of statistically significant differences with an improvement of 5 mm for the calcitonin and a deterioration of 11 mm for placebo ($P = 0.03$) on visual analog scale for ‘back pain’. The results of our study in 2004 were in contrast to several previous trials in the literature reporting subcutaneous calcitonin as effective in treating symptomatic spinal stenosis [1, 2, 7, 9–11]. One potential explanation for this disparity is the formulation of calcitonin that we used. The current research by the authors again does not render rigid answers to this question but repeats the bioavailability rationale for such effects as in our study. Furthermore the bioavailability of intranasally applied calcitonin is rather low and varied. The authors do not show if any measures to standardize the usage of intranasal spray were in effect (i.e. by cleaning, application of the drug in certain position, etc.) It would have been novel to show the evidence that nasal calcitonin resulted in elevation of calcitonin level in the blood. If the nasal calcitonin was not sufficiently delivered into the body, we could categorically reinforce the conclusion that calcitonin does not appear to have a role in conservative treatment of LSS.

In comparison to previous studies with serious methodologic flaws, although Tafazal et al do conduct a randomized study, they however do not answer why previous responders to calcitonin after the wash out period did not see the beneficial effects. Finally, we were of the opinion that our sample size ($n = 55$) chosen based on pilot data may have been insufficient for a rigorous statistical analysis to demonstrate clinically significant differences between the groups. The current authors also have used a smaller sample size of 20 patients in each group to demonstrate outcomes.

In conclusion, we strongly recommend that firstly it is important to investigate the variation in effect via different administration routes (e.g. SQ vs. nasal) and with a longer follow-up. Secondly, other non-operative measures, such as PT, epidural injections, spinal supports, acupuncture, etc. should be explored in well designed randomized trials of clearly defined LSS patients using disease specific outcome measures.

References

1. Eskola A, Alaranta H, Pohjolainen T et al (1989) Calcitonin treatment in lumbar spinal stenosis: clinical observations. *Calcif Tissue Int* 45:372–374
2. Eskola A, Pohjolainen T, Alaranta H et al (1992) Calcitonin treatment in lumbar spinal stenosis: a randomized, placebo-controlled, double-blind, cross-over study with 1-year follow-up. *Calcif Tissue Int* 50:400–403
3. Garfin S, Herkowitz H, Mirkovic S (2000) Spinal stenosis. *AAOS Instr Course Lect* 49:361–374
4. Huth EJ (1986) Irresponsible authorship and wasteful publication. *Ann Intern Med* 104:257–259
5. Jerrells TR (2001) Duplicative publication of data and other ethical issues in publishing of scientific findings. *J Subst Abuse Treat* 20:111–113
6. Katz J, Dalgas M, Stucki G, Lipson S (1994) Diagnosis of lumbar canal stenosis. *Rheum Dis Clin North Am* 20:471–483
7. Onel D, Sari H, Donmez C (1993) Lumbar spinal stenosis: clinical/radiologic therapeutic evaluation in 145 patients: conservative treatment or surgical intervention? *Spine* 18:291–298
8. Podichetty VK, Segal AM, Lieber M, Mazanec DJ (2004) Effectiveness of Salmon calcitonin nasal spray in the treatment of lumbar canal stenosis. *Spine* 29(21):2343–2349
9. Porter RW, Hibbert C (1983) Calcitonin treatment for neurogenic claudication. *Spine* 5:90–93
10. Porter RW, Miller CG (1988) Neurogenic claudication and root claudication treated with calcitonin: a double-blind trial. *Spine* 13:1061–1064
11. Streifler J, Hering R, Gadoth N (1989) Calcitonin for pseudoclaudication in lumbar spinal stenosis. *J Neurol Neurosurg Psychiatry* 52:543–544
12. Tafazal SI, Ng L, Sell P (2006) Randomised placebo-controlled trial on the effectiveness of nasal salmon calcitonin in the treatment of lumbar spinal stenosis. *Eur Spine J* (in press)