A closer look at synovial cysts

TO THE EDITOR: We read with interest the article by Bruder et al.2 (Bruder M, Cattani A, Gessler F, et al: Synovial cysts of the spine: long-term follow-up after surgical treatment of 141 cases in a single-center series and comprehensive literature review of 2900 degenerative spinal cysts. J Neurosurg Spine 27:256–267, September 2017). In this study, the authors presented the clinical data of 141 patients with synovial cysts who were treated surgically between 1997 and 2014. The majority of patients underwent hemilaminectomy, and a small subset of patients were treated with laminectomy or interlaminar fenestration. The facet joint was spared in most cases (63%), while the medial two-thirds and one-third of the facet joint were resected in 30% and 7% of patients, respectively. Based on the study results, 88% of patients with preoperative leg pain were “completely pain free” of leg pain after surgical treatment. Interestingly, the rate of low-back pain declined from 90% preoperatively to 5% postoperatively, and 86% of patients with preoperative low-back pain were back “pain free” after surgery. At 9 years of follow-up, 6% reported back pain.

A word of caution is in order here. This study reports that facet-sparing hemilaminectomy led to complete resolution of low-back pain in 86% of cases, and an absence of both leg and back pain on long-term follow-up. These patients were mostly in their 6th decade of life with facet disease, which led to the synovial cyst herniation. Low-back pain is a complex, multifactorial etiology that is unlikely to be resolved with hemilaminectomy or laminectomy. There is an emerging body of evidence that structural pathology, such as facet joint arthropathy and degenerative disc disease, is the main culprit of low-back pain.1 Although one would argue that the synovial cyst itself is a manifestation of the facet joint degenerative process, surgically removing the cyst without addressing the diseased facet joint, which is the source of back pain, might decrease the severity of low-back pain, but not completely resolve it.2,3 For instance, a larger series by Xu et al. showed that patients treated with laminectomy or hemilaminectomy reported a higher incidence of back pain at 2-year follow-up than patients who underwent fusion (p = 0.002), although the same patient population showed improvement in back and leg pain immediately after surgery.2 Another series of 46 patients (by Weiner et al.) with a longer follow-up time than the current series showed that 28% of patients had low-back pain after an average of 9.7 years postoperatively.4 Therefore, the current conclusion of persistence of back pain in 6% of patients 9 years after surgery is questionable.

Additional factors, including physical, social, psychological, and nonmodifiable genetic ones, could contribute interchangeably, along with structural pathologies, to low-back pain. The multitude of these factors poses a challenge for providers caring for patients with low-back pain. Typically, the presence of mechanical back pain and radiological signs of structural pathology (i.e., widening of the facet joints, hyperintense T2 signal on MRI, collapsed disc with Modic changes, spondylolisthesis) might favor fusion. With this in mind, it is important to recognize that there is no cure, as suggested in Bruden’s study, for low-back pain, even if we were to eliminate the pathological spinal unit with fusion.4,5

Another marked limitation of this study that adds to its lack of statistical insignificance is the small number of follow-up patients (81 patients). Bruden et al. repeatedly stated that their series has the second longest follow-up time. However, their follow-up rate, 57%, was the lowest of all other studies. The authors tried to avoid confronting this limitation by comparing the total number of follow-up patients instead of the percentages, and by stating that they “achieve[d] a reasonable follow-up rate.” This major limitation should have alerted the reviewers of this article at your esteemed journal.

Moreover, the fact that the follow-up process was based on telephone interviews “with structured, questionnaire-based information” raises the following questions: how did the authors assess the patients’ motor function, sensation, and whether the leg pain was residual or new (different dermatome)? More importantly, how was the information about cyst recurrence, the level of recurrence, and rates of delayed fusion obtained?

In their conclusion, the authors stated, “With facet-sparing techniques, the stability of the segment can be preserved.” Although we agree with this statement, it does not hold true for the findings of this study. First, the facet-sparing technique was performed in 63% of the patients while 37% underwent partial facetectomy. Second, the authors did not state how they studied the “segmental instability” given that postoperative imaging was not performed in any of the patients. Finally, a clear distinction must be drawn between “segmental instability” (> 4 mm of motion of the vertebrae on flexion-extension radiographs) and “spondylolisthesis” (vertebral body slippage).3
The authors investigated the presence of postoperative “spondylolisthesis” in the follow-up period, but reported on “segmental instability” in the conclusions. All the previous methodological issues limit the conclusions of this study, and should call into question the relevance of its findings.

In summary, facet arthropathy is a cause of back pain. A hemilaminectomy may be able to remove the cyst and help the patient’s radiculopathy, but we doubt that most neurosurgeons would promise a 94% chance of “back pain free” outcome over the next decade, after a hemilaminectomy, as suggested by this article.

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References

Disclosures
The authors report no conflict of interest.

Response
We read with interest the Letter to the Editor by Ramhmdani and Bydon commenting on our recently published article.

To be clear, assessing the clinical situation via telephone interview is clearly a limitation of our study, as we mentioned in the Limitations of the Study section. We also mentioned the assessment of recurrent cysts and delayed fusion in the Methods section.

Without doubt, we were not able to contact 42% of patients for long-term follow-up and clearly stated the numbers and percentages, as well as the reasons for losses to follow-up in the text and tables. Nonetheless, we were able to obtain data in 81 patients with a mean follow-up period of more than 9 years. This remains the largest series with a follow-up period of more than 4 years (Supplemental Table 1), proving these data to be worthy of publication.

However, when questioning the results and conclusion of our report and focusing on the symptom “low-back pain,” Ramhmdani and Bydon were somehow misled. We never stated in our report that 86% of patients were free of leg or back pain at the time of long-term follow-up, and we never promised a 94% back-pain free rate or even “cure” for low-back pain on long-term bases. In our study, additional low-back pain was reported in 127 patients before the operation, and 86% of those patients were pain free at short-term follow-up after surgery. At the time of long-term follow-up, however, 6% of 81 patients of the follow-up group reported low-back pain, and an additional 22% reported low-back pain under physical stress. As stated in Table 3, the overall rate of back pain in the follow-up group was 28% compared with the 22% back pain rate reported in the 594 cases of the literature in which “back pain” at the time of follow-up was mentioned. Therefore, we absolutely agree with Ramhmdani and Bydon that low-back pain has a “complex, multifactorial etiology,” and its treatment is and will be challenging.

In the end, we did not draw our conclusion from the presence or absence of back pain but from the overall long-term clinical situation, which should be the primary goal in degenerative spine surgery.

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Thinking laterally: beyond transoral decompression for irreducible ADD with os odontodisc

TO THE EDITOR: I read the recently published article by Wu et al.10 (Wu X, Wood KB, Gao Y, et al: Surgical strategies for the treatment of os odontodeum with atlantoaxial dislocation. J Neurosurg Spine 28:131–139, February 2018). The authors have described surgical strategies for treating patients with atlantoaxial dislocation (AAD) with os odontodisc. They classified patients based on reducibility of dislocation. Those with reducible AAD were treated with posterior stabilization alone, whereas those with irreducible AAD underwent transoral decompression followed by posterior stabilization. The posterior stabilization was short segment in most, with the exception of 4 patients in whom more than 2 segments were fused.

The authors have been practicing erstwhile surgical methods and have failed to take notice of contemporary techniques of direct posterior reduction. The surgical procedures for AAD have undergone significant change in the last 2 decades.1,3,6,7 The focus has shifted to the lateral C1–2 joints rather than decompression of the dens. Recent techniques involve manipulation of the C1–2 lateral joints to achieve reduction, followed by C1–2 fusion. In the current era of direct posterior reduction, to preoperatively differentiate reducible from irreducible AAD is to pre-empt the procedural difficulties.1

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