Letters to the Editor

NEUROSURGICAL FORUM

Complications associated with epidural steroid injection

TO THE EDITOR: We read with great interest the recently published article by Kraeutler et al.⁴ (Kraeutler MJ, Bozzay JD, Walker MP, et al: Spinal subdural abscess following epidural steroid injection. *J Neurosurg Spine* 22:90–93, January 2015). The authors presented a case of spinal subdural empyema, which is a rare but serious complication associated with epidural steroid injections (ESIs). We commend the authors' early recognition and prompt treatment of this serious and potentially devastating complication, which led to significant improvement in the patient's neurological status.

The efficacy of ESIs in the management of low-back pain and radiculopathy has been a matter of great controversy. There has been an absence of high-quality evidence to support their repeated use in management of chronic low-back pain with or without radiculopathy. The most recent guidelines from the American Association of Neurological Surgeons/Congress of Neurological Surgeons Joint Guidelines Committee recommend ESIs only as an option for short-term management of low-back pain, with very weak evidence (Level III) supporting their use.⁹ Moreover, a recent double-blinded, randomized controlled trial published in *The New England Journal of Medicine* demonstrated lack of efficacy at 6 weeks in clinical outcome when comparing ESIs with epidural lidocaine injections.³ Despite the lack of evidence for efficacy as a long-term management for chronic back pain due to spinal spondylosis and stenosis, the use of ESIs has increased dramatically in recent years, with an estimated 11 million injections performed in the US annually.¹ While mostly benign, potential rare but serious complications, such as spinal epidural/subdural hematomas, spinal epidural/subdural abscesses, and even cerebral/spinal cord infarctions, have been reported in the literature.^{2,4,7,8,10,11}

Given the limited data on long-term efficacy of repeated ESIs in patients with chronic back pain with or without radiculopathy, as well as small but potential chances of untoward complications, use of repeated ESIs in patients with chronic low-back pain should preferably be avoided. Even though an ESI has been shown to provide short-term relief in patients with back pain from stenosis, about a quarter of patients may not respond to ESIs with the first 2 injections; these patients should not be subjected to multiple subsequent injections given that there are small but

real chances of significant complications,⁵ which cannot be ignored. In fact, in patients with obvious spinal stenosis on imaging and minimal or no response to the first ESIs, surgical options has been shown to result in good clinical outcome and should be timely entertained.⁶

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DISCLOSURE

The authors report no conflict of interest.

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Response

No response was received from the authors of the original article.

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Atlantoaxial instability and Chiari malformation

TO THE EDITOR: I found the article by Goel² (Goel A: Is atlantoaxial instability the cause of Chiari malformation? Outcome analysis of 65 patients treated by atlantoaxial fixation. *J Neurosurg Spine* 22:116–127, February 2015) to be very interesting and controversial.

In this paper, Prof. Atul Goel, one of the world leaders in craniocervical junction surgery, proposed that Chiari malformation (CM) (with or without concomitant basilar invagination) is secondary to atlantoaxial instability.² He also proposed that C1–2 posterior instrumented fusion should be performed alone and without concomitant posterior fossa decompression, a well-accepted treatment in many referral centers around the world that routinely treat CM. Considering the controversial content of the article, I would like to make some comments about its content.

1) If this theory is true, why did tonsillar herniation or syringomyelia not develop in patients with other atlantoaxial instabilities? As an example, patients with chronic atlantoaxial traumatic instability and os odontoideum, among other atlantoaxial diseases, did not have tonsillar herniation. Atlantoaxial subluxation in the setting of rheumatoid arthritis is quite common, and tonsillar herniation and syringomyelia are not mentioned as common radiological findings.

2) Spontaneous regression of syringomyelia in CM has been reported. How is it possible to explain this in the setting of atlantoaxial instability?^{3,6,9}

3) There are many studies addressing reduction of the posterior fossa volume in patients with CM, as well as an association with platybasia and a shorter clivus.^{1,4,5,7,8} In his paper, Prof. Goel suggested that this theory does not seem to be valid. Why? The article did not explain this properly.

4) Can we put all congenital craniocervical malformations and their different forms of presentation together and treat all of them in the same way, with atlantoaxial fixation? Should patients with clear craniocervical instability and severe forms basilar invagination and bone anomalies be treated exactly like patients with mild to moderate cervical pain secondary to a 5-mm tonsillar herniation without syringomyelia? In my humble opinion, I do not think this make sense.

Additionally, I did not understand how a patient with normal findings on flexion and extension atlantoaxial sagittal CT scans or MR images, as well as normal facet joint morphology, can have an “occult” instability.

5) What is the definition of atlantoaxial instability in this context?

6) Finally, and probably the most important point, many articles have described syringomyelia regression and clinical improvement of patients with tonsillar herniation after posterior fossa decompression, with or without duraplasty.^{1,5,7,8} I believe that performing atlantoaxial fixation instead of a historically accepted safe and efficient procedure such as posterior fossa decompression in a patient with CM without a clear and evident craniocervical or atlantoaxial instability must involve several ethical issues until further evidence is available.

I thank the *Journal of Neurosurgery* very much for the opportunity to try to clarify these points. I congratulate Prof. Atul Goel for his new and challenging theory for explaining congenital craniocervical malformations. I hope that he can answer this letter with his remarkable comments and unique view of craniocervical junction pathologies.

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DISCLOSURE

The author reports no conflict of interest.

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Response

Dr. Joaquim has raised certain important issues regarding my hypothesis about the relationship of CM with atlantoaxial instability. The understanding of the etiopathogenesis of a number of “anomalies” at the craniovertebral junction is still at the speculation level. No definite genetic disorder, nutritional deficiency, or systemic cause has been identified. Error in embryogenesis has been implicated as the cause of disordered structural formation. The possible genesis may be multifactorial.

My article proposes that CM is a secondary effect to primary atlantoaxial instability.⁴ The instability is longstanding and probably occurs early in infancy or during later stages of fetal life. Birth-related injuries and malnutrition seem to be an important predisposing cause. The beginning of the structural malformation probably occurs during the period when organogenesis is not complete and the process of physical formation can be molded according to the functional demands. The enormity of the natural processes that happen during the healthy period of the organism, and evolve during “disease,” are truly awe inspiring. Our article that was published in *Journal of Neurosurgery: Spine* in 2009 suggested that a number of musculoskeletal alterations associated with basilar invagination could be reversed following surgery that involves only stabilization of the atlantoaxial joint.¹¹ A short neck can become normally long, and torticollis can reverse. Secondary spondylotic changes and bone fusions have a potential to reverse. The CM seems to be Nature’s innovative formation that acts as an airbag and positions the tonsils in a strategic location that prevents pinching of critical neural structures between bones.⁴ Syringomyelia also seems to be protective self-destruction of neural tissues that works in the larger interest of the patient and assists in neutralizing the spinal and cranial pressures.⁶ The very fact that CM can reverse and a syrinx can disappear after surgery that involves only atlantoaxial fixation validates the hypothesis. The clinical result of surgery settles the issue. It is unclear as to why CM is not associated with all kinds of atlantoaxial instability. The timing of initiation of the dysgenesis is probably the defining factor. Traumatic atlantoaxial dislocations (acute or chronic) and dislocations related to rheumatoid arthritis, as mentioned by Dr. Joaquim, occur late in life when the structural formation cannot be molded. The issue of spontaneous regression of tonsils and its relationship to atlantoaxial instability will have to be evaluated on a case-by-case basis.

We were the first to evaluate the clinical significance of a small posterior cranial fossa and its relationship with CM and its importance in formulating the treatment protocol.⁸ We recommended foramen magnum decompression in specific groups of patients. We now identify that the posterior fossa is not tight in these cases. Evidence of atrophy of the cerebellum and vermis is frequently observed in the presence of CM.⁹ In our article we mentioned that the clinical improvement following foramen magnum decompression may be akin to deflating the airbag.⁴ Although widely and popularly performed, such an operation may be effective in the postoperative period, but in the long run it may be counterproductive.

It may indeed be possible that a number of clinical enti-

ties encountered in the craniovertebral junction are related to atlantoaxial instability. Despite the fact that facets are the only true joints of the spine, the focus of attention has been the odontoid process in the craniovertebral junction and discs in the rest of the spine.^{1,2,5,7,10} The parameter of evaluation of instability has always been the odontoid process. The evaluation of patterns of “facetal” instability as presented by us opens up a space for fresh understanding of the region and the subject.³ The issue of central or axial instability seems to be an interesting concept and a real phenomenon.

The rapidity in the evolution of understanding of various issues of craniovertebral junction epitomizes the scientific and medical revolution that has taken place in the last few decades hallmarked by growth of computer technology. Newer forms of treatment and alternative strategies will certainly evolve. It may be pertinent to sit down and think about the possible useful implications of any proposed thought.

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