Deformity surgery


Age has been proposed as a well-established predictive factor for major postoperative morbidities in long-segment thoracolumbar spinal surgery. Complication rates in the elderly are relatively higher than in the younger population; therefore, careful patient selection as well as good clinical judgment about the best approach is mandatory before undertaking long-segment thoracolumbar spinal surgery in older patients. Acosta et al. conducted a retrospective study focused on the comorbidities of very elderly patients who underwent long-segment (5 or more levels) fixation for spinal deformity. They showed that patients 75 years and older undergoing major spinal correction surgery have an overall perioperative complication rate of 62%, with an older age augmenting the incidence of complications, and a long-term postoperative complication rate of 52%. Elderly hypertensive patients are 10 times more liable to incur a major postoperative complication.

There were 2 minor concerns about their study. First, the study focused on very elderly patients, who are more likely to have a higher incidence of osteoporosis. Bone quality is an important factor that influences the stability of posterior spinal implants. However, the authors did not discuss the preoperative evaluation of bone mineral density (BMD). Impairment of bone quality would lead to a high risk of screw pullout or spinal fractures at the adjacent level. In selecting long-segment spinal deformity surgery, BMD is a fundamental preoperative evaluation item for assessing the risk of instrumentation-related complications. Importantly, osteoporosis requires optimal medical management to prevent further collapse of vertebrae. Second, their study addressed radiological outcome. We would like to recall one important issue focused on clinical neurological outcomes. In fact, long-segment spinal fusion often leads to back pain, which stems from back muscle traction-related degeneration.

Despite these concerns, their study has provided a very important reference for the assessment of morbidity and mortality after long-segment spinal deformity surgery in patients 75 years and older. Spinal surgeons should take great precautions in the selection and care of very elderly patients undergoing long-segment spinal fusions. Additional large-scale, multicenter prospective studies will further illuminate more pearls of high-quality spinal care for patients.

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Disclosure

The authors report no conflict of interest.

References


Response: We thank Dr. Hueng and colleagues for reviewing our article. One of our goals was to evaluate an important population group, those 75 years of age and older, and to study complication rates following long-segment instrumented spinal fusions. We hope that our study provides clinicians who are treating the very elderly a framework to aid in patient selection for surgical correction of deformity.

Bone health and quality and the impact of smoking, osteopenia, and osteoporosis on long-term outcomes for patients undergoing deformity correction are important considerations for the surgeon. It is known that poor bone health can lead to instrumentation failure, including pedicle screw pullout. Although we did not directly evaluate the effect of osteoporosis and osteopenia and the risk of perioperative complications, indirect assessment

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can be extrapolated. Osteoporosis is a known risk factor for the development of vertebral compression fractures. Vertebral compression fracture occurred in 2 patients (1 treated with vertebroplasty, and the other treated conservatively), but we did not consistently evaluate preoperative BMD in all patients. Proximal junctional kyphosis is a known condition following instrumented fusions; however, BMD was not a statistically significant factor for its development. More spinal surgeons are obtaining BMD as part of the preoperative assessment when considering an operation.

Radiographic and clinical outcomes following spinal surgery are becoming standards for determining the best methods for treatment. Prospective multicenter studies are needed for looking at radiographic and health-related quality of life outcomes for patients 75 years and older to optimize care. Despite the limitations inherent to our study because of its retrospective nature, we hope that all physicians treating spinal disorders will consider surgery as part of potential management and will not exclude it based on patient age.

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References


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Critical illness polyneuropathy/critical illness myopathy and acute motor-sensory axonal neuropathy

To The Editor: We read the case report by Cheng et al. with great interest (Cheng J, Kahn DE, Wang MY: The acute motor-sensory axonal neuropathy variant of Guillain-Barré syndrome after thoracic spine surgery. Case report. J Neurosurg Spine 15:605–609, December 2011). The authors described the case of an acute motor-sensory axonal neuropathy (AMSAN) variant of Guillain-Barré syndrome (GBS) after surgery. Although GBS has been reported to occur in postoperative settings, the commonly encountered types of neuromuscular dysfunction in the intensive care unit (ICU)—that is, critical illness polyneuropathy (CIP) and critical illness myopathy (CIM)—should be considered in the diagnosis, because CIP can develop as early as within 3 days after sepsis onset, as happened in the authors’ patient. Their report did not provide more detailed clinical data contributing to the diagnosis, such as information on 1) any history of infection before the operation; 2) the normal facial grimace as facial muscle unaffected; 3) the use of neuromuscular blocking agents, corticosteroids, aminoglycoside antibiotics, catecholamines, and parenteral nutrition potentially related to CIP/CIM in different clinical processes; 4) the level of creatine kinase in serum; and 5) muscle biopsy results. In the ICU, there are 2 kinds of neuromuscular abnormalities. The first kind refers to the neuromuscular disease, such as GBS, as a reason for admission to the ICU. In the second, flaccid paralysis is acquired in the ICU without preexisting neuromuscular disorders like CIP/CIM. Although rare, GBS is sometimes difficult to differentiate from CIP/CIM.

Both CIP and CIM are frequent complications of critical illness, involving the motor and sensory axons of the peripheral nerve system and muscle. In the ICU, CIP and CIM present as flaccid and symmetric (including respiratory muscles) weakness and/or sensory loss. Studies have shown that CIP and/or CIM develop in approximately 70% of patients with sepsis or systemic inflammatory response syndrome, 60% with acute respiratory distress syndrome, and up to 100% of patients with multiple organ failure. Both CIP and CIM prolong weaning from mechanical ventilation and physical rehabilitation.

The Medical Research Council grading system (sum score) is used as an initial diagnostic physical examination of muscle force in awake patients with suspected CIP/CIM. Further technical investigations, including serum creatine kinase level, electromyography (EMG), and muscle biopsy, may provide more reliable information in making a diagnosis. Diagnosing CIP and CIM is occasionally difficult because of either the preexisting disorder or complications arising when the hospitalization causes limb and respiratory muscle weakness. When critically ill patients develop flaccid paralysis and EMG shows evidence of motor and sensory axonal polyneuropathy, CIP can be diagnosed if limb weakness or difficulty weaning patients from a ventilator after nonneuromuscular causes, such as heart and lung diseases, have been excluded. Guillain-Barré syndrome may sometimes occur under the same conditions, so it is important to identify the syndrome because of its response to specific immunomodulatory treatment. An axonal damage form of GBS may be more difficult to distinguish from CIP because of the similar EMG sign. Criteria for differentiation are specified in Table 1. Although clinical presentations and EMG studies do not provide supportive points for discriminating between CIP and GBS, CSF analysis
might be more useful. Nevertheless, CSF protein concentrations in patients with GBS are often normal in the 1st week after the onset of GBS but increase in more than 90% of patients at the end of the 2nd week. In the authors’ case, the dynamic increased CSF protein concentrations are mostly resected, the CSF leak from this operation was not easy to avoid. Therefore, the increased protein in the CSF will last for a long time, so it might cause a confused diagnosis or exclusion of CIM.

We suggest considering a diagnosis of CIP/CIM instead of GBS in the authors’ reported case, if the further clinical data they provide are inclining to CIP/CIM. After all, the most likely cause of limb muscle weakness in the ICU is CIP/CIM. According to Koch et al., CIM is more frequent than CIP, and most patients with CIP feature concomitant CIM. In this regard, detection of serum creatine kinase and muscle biopsy may be helpful in the diagnosis or exclusion of CIM.

**Disclosure**

The authors report no conflict of interest.

**References**


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**TABLE 1: Criteria for differentiation between CIP and GBS**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>CIP†</th>
<th>GBS‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>precipitating factor</td>
<td>sepsis/SIRS, organ failure</td>
<td>minor infection, trauma</td>
</tr>
<tr>
<td>clinical presentation</td>
<td>onset of disorder after ICU admission; often characterized by fairly symmetric limb muscle weakness &amp; maintaining cranial nerve function including unaffected facial nerve &amp; muscle</td>
<td>onset of disorder before ICU admission; before op, infection precedes onset of progressive weakness &amp; sensory disturbances</td>
</tr>
<tr>
<td>CSF analysis</td>
<td>normal except albumin increased by surgical intervention</td>
<td>albuminocytologic dissociation</td>
</tr>
<tr>
<td>electrophysiology</td>
<td>1) axonal motor &amp; sensory polyneuropathy; compound motor &amp; sensory nerve action potentials: reduction of amplitude w/o changes in latency, w/o changes in conduction velocity; fibrillation &amp; positive sharp waves</td>
<td>1) demyelinating polyneuropathy or unresponsive nerves, abundant spontaneous activity; 2) axonal motor &amp; sensory polyneuropathy</td>
</tr>
<tr>
<td>biopsy</td>
<td>primarily axonal degeneration of distal peripheral nerves w/o inflammation</td>
<td>primarily demyelinating process w/ inflammation, or motor &amp; sensory axonal degeneration, or motor axonal degeneration only</td>
</tr>
<tr>
<td>treatment</td>
<td>treat sepsis</td>
<td>plasmapheresis, intravenous immune globulin</td>
</tr>
<tr>
<td>clinical course</td>
<td>recovery</td>
<td>75% have complete recovery, 30% require ICU admission</td>
</tr>
</tbody>
</table>

* SIRS = systemic inflammatory response syndrome.
† See Algahtani et al., 2009, and Green, 2005.
‡ See Algahtani et al., 2009; Green, 2005; Hughes and Cornblath, 2005.
CSF is considered supportive of GBS. Wang and colleagues raise the valid point that elevated CSF protein has been reported in patients with meningiomas and that CSF leakage may have contributed to our patient’s initial CSF profile. However, the fact that her CSF protein level continued to increase in the face of a decreasing red blood cell count, symptom progression, and after meningioma removal makes this an unlikely contributing source of CSF protein.

The patient did not receive aminoglycosides or total parenteral nutrition. Muscle biopsy was not performed. Nor would a myopathy explain our patient’s length-dependent sensory impairment.

We thank Dr. Wang and colleagues for this opportunity to expand upon the differential diagnosis of acute muscular weakness and sensory dysfunction in the ICU setting. However, the diagnosis of AMSAN, rather than CIP/CIM, is supported by the features of facial, bulbar, and appendicular weakness; length-dependent sensory dysfunction; and the significance of the diagnostic testing as discussed above.

References


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Accessory articulation

To The Editor: We read with great interest the paper of Mahato (Mahato NK: Disc spaces, vertebral dimensions, and angle values at the lumbar region: a radioanatomical perspective in spines with L5–S1 transitions. Clinical article. J Neurosurg Spine 15:371–379, October 2011).5

In this study the author performed a detailed morph-
ometric analysis of the radiographic dimensions of intervertebral disc spaces, vertebral bodies, pedicles, vertebral canal, and transverse processes of lumbar vertebrae in patients with lumbosacral transitional states and low-back pain (LBP), and compared such findings with data collected from a control group of patients with LBP but without lumbosacral anomalies.

The study’s results indicated that several morphometric parameters from the patients with abnormal L5–S1 accessory articulations were significantly different from those of the control group. Furthermore, as mentioned by the author, some previous studies have suggested that such anatomical differences do lead to significant differences in the functional biomechanics of the lumbosacral spine, such as restricted rotational and bending movements below the L5–S1 transition and a consequent dys-equilibrium in load bearing at the L5–S1 junction.\(^6,7\)

Although it seems clear from Mahato’s study that L5–S1 transitional states significantly impact the overall functional dynamics of the lumbosacral spine, determining the exact cause of LBP in each patient presenting with lumbosacral transitional states remains a challenging clinical task.

In relation to the several possible causes of LBP in such a population, it is very important to differentiate between a specific group that presents with facetectomy pain related to the abnormal contact of the “neoarticulation” of the abnormal transverse process with the sacrum, and a group that presents with LBP consequent to other anatomical changes occurring in lumbosacral transitional states, such as discogenic pain related to degenerative disc disease in the adjacent level, extraforaminal stenosis secondary to nerve root compression by the enlarged L5–S1 transverse process, and facet pain from other zygoapophyseal articulations besides the abnormal lumbosacral joint.

We previously proposed a simple algorithm in order to properly identify this subgroup of patients with lumbosacral transitional states in which the transverse mega-apophysis may be implicated in the etiology of LBP (Fig. 1).\(^2\) In our experience anesthetic blocks have proven to be very useful as therapeutic tests to confirm the possible relation between LBP and the abnormal L5–S1 articulation. In patients who present improvement in such blockages, conventional radiofrequency neurolysis may be offered as an initial, less invasive intervention that may provide significant pain relief and facilitate future physical rehabilitation therapies. In such a subpopulation, if symptoms recur, we have also demonstrated that resection of the transverse mega-apophysis at the symptomatic side can be used as a second-line therapy that confers excellent results at the long-term follow-up.

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**Fig. 1.** Proposed diagnostic-therapeutic algorithm for evaluation and treatment of patients with lumbosacral transitional states and LBP.
a lumbosacral transitional state (Castellvi Type IIa) in which the abnormal neoarticulation of the elongated left L-5 transverse process with the sacrum demonstrates evident signs of advanced degeneration and pseudarthrosis.

The aforementioned diagnostic-therapeutic algorithm is especially important in patients presenting with lumbosacral transitional states classified as Castellvi Types II–IV. Such patients represent those in whom it is possible to demonstrate the real existence of an abnormal “neoarticulation” between the enlarged transverse process and the sacrum (Fig. 2). Such a subgroup of patients has been classically described as harboring the so-called Bertolotti syndrome. Nevertheless, some authors also include patients with Castellvi Type I lumbosacral transitional states in such a syndrome, defending the position that short and broad iliolumbar ligaments may make the L5–S1 disc in such a syndrome, defending the position that short and broad iliolumbar ligaments may make the L5–S1 disc space more rigid and potentially destabilize the L4–L5 level, leading to the same biomechanical consequences observed in the complete forms.

As recently mentioned by a renowned academic spine surgeon: “Ultimately, the determination of whether a manuscript is publishable comes down to 2 criteria: 1) level of interest and 2) archival value.” In the case of Mahato’s study, the high prevalence of lumbosacral transitional states as well as its common association with LBP gives this publication a very high level of interest for the whole spine surgery community. Furthermore, the thorough characterization of the several morphometric parameters associated with lumbosacral transitional states confers on this study an incommensurable archival value for the scientific literature and future studies on the issue.

In summary, we congratulate the author for such extensive and laborious work, which greatly enriched our anatomical knowledge regarding lumbosacral transitional states as well as laid the foundations for further biomechanical studies investigating the functional biomechanical changes generated by such morphometric abnormalities. Finally, this type of basic science knowledge is of paramount importance in providing spinal surgeons with a better understanding of the etiology of LBP in such a population as well as paving the way for future clinical studies aimed at establishing a more individualized therapeutic strategy for each patient with better long-term functional outcomes.

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Neurosurgical forum

Disclosure

The authors report no conflict of interest.

References

3. Benzel EC: Anatomic studies and their relevance; publication criteria, level of interest, and archival value. World Neurosurg 74:268–269, 2010

RESPONSE: It is very encouraging to read the letter regarding my article. I appreciate the clinicians’ acknowledgement of the significance of the basic and fundamental morphological features that distinguish atypical spines from typical ones.

Morphological and hence functional/biomechanical differences exist between the spines of the two sexes, between normal and “atypical” spines, and thus, possibly, between spines that are more susceptible to LBP and those that are dynamically more resilient to abnormal degeneration of the components in a motion segment. Examination of neuromuscular and biomechanical adaptations in “transitional spines” could go a long way in understanding the pathophysiolo of back pain situations and in “enabling a more individualized approach with better functional outcomes” for treatment strategies, as suggested by the observers.

I am thankful for recognition of the fact that these morphological variations may be helpful in initiating future investigations of physiological aspects involving lumbosacral transitional abnormalities.

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