



Review

Iatrogenic neurologic deficit after lumbar spine surgery: A review



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ABSTRACT

Iatrogenic neurologic deficits after lumbar spine surgery are rare complications, but important to recognize and manage. Complications such as radiculopathy, spinal cord compression, motor deficits (i.e. foot drop with L5 radiculopathy), and new onset radiculitis, while uncommon do occur. Attempts at mitigating these complications with the use of neuromonitoring have been successful. Guidance in the literature as to the true rate of iatrogenic neurologic deficit is limited to several case studies and retrospective designed studies describing the management, prevention and treatment of these deficits. The authors review the lumbar spinal surgery literature to examine the incidence of iatrogenic neurologic deficit in the lumbar spinal surgery literature.

An advanced MEDLINE search conducted on May 14th, 2015 from January 1, 2004 through May 14, 2015, using the following MeSH search terms “postoperative complications,” then subterms “lumbar vertebrae,” “treatment outcome,” “spinal fusion,” and “radiculopathy” were included together with “postoperative complications” in a single search. Postoperative complications including radiculopathy, weakness, and spinal cord compression were included. The definition of iatrogenic neurologic complication was limited to post-operative radiculopathy, motor weakness or new onset pain/radiculitis.

An advanced MEDLINE search conducted on May 14th, 2015 using all of the above terms together yielded 21 results. After careful evaluation, 11 manuscripts were excluded and 10 were carefully reviewed. The most common indications for surgery were degenerative spondylolisthesis, spondylosis, scoliosis, and lumbar stenosis. In 2783 patients in 12 total studies, there were 56 patients who had reported a postoperative neurologic deficit for a rate of 5.7. The rates of deficits ranged from 0.46% to 17% in the studies used. The average rate of reported neurologic complications within these papers was 9% (range 0.46–24%). Thirty patients of a total of 731 (4.1%) had a new onset neurologic injury after anterior lumbar interbody fusion or lateral lumbar interbody fusion. Thirty-seven out of 2052 (1.9%) patients had a neurologic injury after posterior decompression and fusion. Screw malposition was responsible for 11 deficits.

Spinal surgery for lumbar degenerative disease carries a low but definite rate of neurologic deficits. Despite the introduction of neuromonitoring, these complications still occur. Interpretation of neurologic injury rates for lumbar surgery is limited by the few prospective and cohort-matched controlled studies. Likewise, most injuries were associated with the placement of instrumentation despite the type of approach.

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1. Introduction

Iatrogenic neurologic deficits after lumbar spine surgery are rare complications, but are arguably the most feared complication of spinal surgery and are important to recognize and manage. Complications such as radiculopathy, motor weakness, spinal cord compression, and postoperative neuropathic pain, while uncommon, do occur. Early work in adult and pediatric spinal deformity by Bridwell and colleagues provide the first detailed reports of iatrogenic neurologic deficit with a rate of 0.4% [1].

Iatrogenic neurologic deficits may occur by a number of mechanisms. Arguably, the most common mechanism is due to mechanical compression, an example of which is by way of an expanding, space-occupying process such as a postoperative hematoma, or via compression by instrumentation. Another possible means of direct compression can also occur as a result of deformity corrective measures resulting in neural element compression. Less commonly, distraction of the spinal cord can occur from overcorrection of sagittal balance, column shortening, or column lengthening maneuvers [1]. Also hypothesized is a vascular mechanism of spinal cord injury due to thrombosis of a segmental vessel resulting in neurologic deficit [1,2].

Although rare, Bridwell and coinvestigators find that hyperkyphotic patients undergoing anteroposterior deformity correction are at a relatively higher risk of postoperative neurologic deficit from a purely vascular origin. This is likely due to a disruption of blood flow to the thoracic spinal cord through segmental arterial feeders from the aorta [1]. Intraoperative deficits that spinal surgeons should be aware of have been reviewed which include motor and sensory deficits [1,3]. Attempts at mitigating these complications with the use of intraoperative monitoring via somatosensory evoked potentials (SSEPs) have been successful [4–6].

The majority of the aforementioned literature highlighting iatrogenic neurologic deficits has historically been in the field of scoliosis. The authors review the literature in attempt to highlight the incidence of iatrogenic neurologic deficit in lumbar spinal surgery.

2. Methods

A MEDLINE search for pertinent literature was conducted on May 14th, 2015 from January 1, 2004 until May 14, 2015 using studies in English along with the following MeSH search term: 'postoperative complications.' This was combined with the sub-terms 'lumbar vertebrae,' 'treatment outcome,' 'spinal fusion,' and 'radiculopathy' in a single search. Studies delineating the outcomes, nature, cause, and follow-up of immediate postoperative complications for lumbar spinal surgery techniques were considered. An iatrogenic neurologic deficit was limited to post-operative radiculopathy, motor weakness or new onset pain/radiculitis.

The search results were evaluated by two authors (K.W., G.G.) to determine if the papers were pertinent to postoperative complications encountered in lumbar spinal surgery for common degenerative spinal indications. Articles that were unclear on the

initial search were included for further review. After a detailed review of the abstract, individual manuscripts were evaluated for final inclusion in the data analysis and the bibliographies were reviewed for any additional studies.

2.1. Inclusion criteria

The main sources of literature reviewed were retrospective and prospective studies published after 2004. Papers specifically delineating postoperative radiculopathy, spinal cord compression, weakness, and radiculitis were included as defined above. Complications were defined by a 90 days postoperative window, a time period commonly defined in numerous complication studies [7–10]. Relevant papers were then identified and selected by the authors for inclusion (K.W., G.G.). All articles were reviewed for inclusion by the remaining authors.

2.2. Exclusion criteria

Among excluded studies were case reports, case series with less than ten subjects, review articles, and papers published prior to 2004. Additional studies that were excluded were case series that did not explicitly delineate the nature of the complications or did not track neurologic complications. All animal or lab research studies, non-English publications, and other articles deemed irrelevant to the study topics of interest by two authors (K.W., G.G.) were excluded. Literature review was conducted in accordance with PRISMA guidelines for reporting of systematic reviews [11].

3. Results

Twenty-one papers were identified in a PUBMED search with the aforementioned parameters evaluating complications of lumbar spinal surgery. After careful evaluation by the authors, 12 manuscripts were included. The most common indications for surgery were degenerative spondylolithesis, spondylosis, scoliosis, and lumbar stenosis (Fig. 1).

In 2783 patients in 12 total studies, there were 56 patients who had reported a postoperative neurologic deficit for a rate of 5.7% (Table 1). The average rate of reported neurologic complications within these papers was 9% (range 0.46–24%). Thirty patients of a total of 731 (4.1%) had a new onset neurologic injury after anterior lumbar interbody fusion or lateral lumbar interbody fusion (ALIF, LLIF) [12–16]. Across the individual reports, 2 patients had new-onset radiculopathy resulting in motor weakness after LLIF [16], 2 after ALIF [13], and 26 had immediate radiculopathy with a motor deficit after LLIF in another report [14,15].

Thirty-seven out of 2052 (1.9%) patients had a neurologic injury after posterior decompression and fusion. Screw malposition was responsible for 11 deficits [17–19]. Eleven patients had radiculitis after placement of BMP for fusion [20]. Nine patients had radiculopathy or spinal cord injury due to placement of grafts, sublaminar wires or deformity correction [12,21,22]. There was one patient

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