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Common surgical complications in degenerative spinal surgery

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Abstract

The rapid growth of spine degenerative surgery has led to unrelenting efforts to define and prevent possible complications, the incidence of which is probably higher than that reported and varies according to the region of the spine involved (cervical and thoracolumbar) and the severity of the surgery. Several issues are becoming progressively clearer, such as complication rates in primary versus revision spinal surgery, complications in the elderly, the contribution of minimally invasive surgery to the reduction of complication rate. In this paper the most common surgical complications in degenerative spinal surgery are outlined and discussed.

Key words: Spine surgery; Complication; Failed back surgery; Instability; Disc herniation

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INTRODUCTION

Spine surgery has grown exponentially over recent decades, with fusion performed for degenerative conditions comprising the lion's share[1]. A recent evidence based review of the literature reported the overall rate of reported complications to be 16.4%[2]. The focus of this particular paper will be those complications that are related to the surgical operation; general medical complications and surgical wound infection are not included (Table 1). The conditions mentioned here are outlined briefly but in reasonable detail; a more elaborate report would be beyond the scope of this paper.

DURAL TEARS

Dural tears happen accidentally during spine surgery. The reported incidence varies from 15.9% in revision surgery[3] to 3.5% in primary lumbar discectomy[4]. These tears are usually the result of direct trauma or laceration, with the Kerrison punch being the instrument most commonly implicated[5]. Intraoperative technical difficulties that appear to predispose to accidental durotomies are dural scarring, adhesions and fibrosis, particularly in revision surgery, an eroded and thin dura as seen in long-standing spinal stenosis, and large disc herniations making dural retraction and nerve root dissection difficult[6].

When recognized intraoperatively, dural tears need to be made watertight to prevent cerebrospinal fluid (CSF) leaks. This is usually accomplished by direct suturing and/or the use of fibrin glue, in addition to muscle or fat graft to cover the area of the tear[3-5,7]. In a large retrospective series, primary repair was successful in the majority of cases, with only 1.8% requiring reoperation for a second defect repair[3]. Similarly, results from the Spine Patient Outcomes Research Trial (SPORT) study

show that incidental durotomy, although associated with increased operative time, blood loss and inpatient stay, does not impact long-term clinical outcome[8-10]. If unrecognized however, these tears can have significant consequences, such as CSF leakage and/or the development of fistulas or pseudomeningoceles. CSF leaks present with headache, nausea, vomiting, and/or photophobia as soon as patients assume an upright posture after surgery[7]. A pseudomeningocele is a CSF-filled cyst that develops from the dura tear. In addition to symptoms related to CSF leakage, compression from the cyst may also result in back pain or even nerve root compression[11,12]. The clinical diagnosis may be confirmed by magnetic resonance imaging (MRI) or computed tomography (CT) myelography. Treatment consists of bed rest, epidural blood patch or fibrin glue, percutaneous or open placement of subarachnoid drain and open direct repair of the dural tear[7,11,12]. As the incidence of iatrogenic CSF fistulas or pseudomeningoceles is between 0.02%-2%, there is limited available evidence on the long term outcome of patients presenting with this complication[7,11,12]. Nevertheless, it seems that open direct dural repair as soon as the dural tear is diagnosed provides the best outcomes[7].

RECURRENT DISC HERNIATION

A recurrent disc herniation is defined as the presence of herniated disc material at the same level and site in a patient that has experienced a pain free interval after discectomy. The reported incidence varies between 5%-23%[13-16]. The only risk factors that have consistently shown a strong association are diabetes mellitus[17,18] and the shape and size of the herniation[19]. Symptomatic recurrent herniations are much less common than radiographic ones (10.2% *vs* 23.1%)[16]. So, care must be taken before attributing the recurrence of low back pain or nerve root symptoms to the herniation. In addition, imaging of the post-operative spine can be difficult to evaluate. A mass lesion at the previously operated level should be differentiated between pseudomeningocele, scar tissue and recurrent disc herniation. Gadolinium-enhanced MRI appears to be the imaging modality of choice in such patients[14,20,21], although intraoperative findings are not in agreement with imaging results in up to 33% of cases[22]. Once the diagnosis is made, treatment options are similar to primary herniations, i.e. conservative (pharmacological modalities, physiotherapy) or surgical. Although revision surgery on the spine is generally associated with poorer outcomes and higher rates of complications, repeat discectomies appear to be an exception, with most authors reporting results similar to those of primary discectomies[15,22-24].

INSTABILITY

Clinical spinal instability is defined as the loss of the spine's ability to maintain its patterns of displacement under physiologic loads. There is no initial or additional neurologic deficit, no major deformity, and no incapacitating pain[25]. Causes of instability are degenerative[26,27] erosion of structures by neoplastic disease[28], trauma[29], spondylolisthesis[30] and iatrogenic (post-laminectomy)[31,32]. In the post-operative patient, instability is most commonly seen after laminectomy without fusion, although even simple discectomy may be complicated by this condition. Clinically, patients may present with low back pain with or without radicular symptoms. Radiographic criteria for spinal instability include translation and angulation of one vertebra relative to another in standing and in flexion-extension radiographs, with Posner's radiographic criteria showing the best correlation with clinical findings and surgical outcomes[25,33,34]. The treatment of post-operative spinal instability is either bracing or instrumented spinal fusion, with surgery exhibiting superior results[34,35].

Table 1 Surgical complications in spinal surgery

Complications
Dural tears
Instability
Junctional kyphosis
Recurrent disc herniation
Pseudarthrosis (non-union)

PSEUDARTHROSIS (NON-UNION)

Pseudarthrosis refers to a failure in osseous union of the intended spinal fusion. Although pseudarthrosis is not always correlated with symptoms or poor results[36,37], most authors agree that a solid fusion results in better clinical outcomes and certainly mitigates any need for reoperation[38-40]. Radiographic confirmation is required to make the diagnosis; signs include a cleft in the fusion mass, failure of incorporation of bone graft, progressive resorption of bone graft, loosening and/or breakage of implants and progressive deformity[41]. Pseudarthrosis can be further graded by the Lenke classification, in the case of posterolateral fusions[42], or by the Brantigan, Steffee, Fraser classification, in cases where PLIF cages are used[43]. Radiography however is dreadfully unreliable in detecting non-union (its accuracy ranges from 82%-68%[44,45]) when compared to surgical exploration. Flexion-extension views may be helpful in detecting instability in the fused segments, although their value in the lumbar spine has been questioned[46,47]. Helical CT scanning has demonstrated better accuracy[48,49] although surgical exploration remains the "gold standard"[50]. When the diagnosis has been made, the decision to operate or not should be made on an individual basis. A period of close observation, during which bracing and activity limitation are employed is certainly reasonable early on, in the hope that delayed union, rather than non-union, will ultimately occur. In the symptomatic patient who shows evidence of pseudoarthrosis later on, revision surgery is warranted. It has been shown that pseudoarthrosis repair can lead to improved clinical results[39,44], although this revision surgery carries a significant risk of recurrent non-union and a persistently poor outcome[39,40,51]. When surgically treat-

ing pseudarthrosis, it is important to remember that better graft material than that used in the index procedure should be used in an optimized environment. This means aggressive removal of fibrous tissue, extensive decortication where appropriate, use of autologous bone, (preferably iliac crest), use of biological modifiers such as electrical stimulation or BMP, replacement of implants when anchorage is questionable, conversion to circumferential fusion whenever possible and if necessary, extension of the fusion and correction of alignment[52,53].

JUNCTIONAL KYPHOSIS

Junctional kyphosis can occur at either end of an instrumented spinal fusion as a result of the increased mechanical demands in the zone adjacent to the fusion. In its strict definition, this occurs when the sagittal Cobb angle between the last instrumented vertebra and two vertebrae further away from this is greater than 10° or when postoperatively there is an increase in the same angle by ⩾ 10°[54]. Incidence appears to be greatest at the proximal end of long fusions, with reported rates ranging from 26%-43%[55-57] while distal junctional kyphosis occurs in 21.7%-30.2% of patients overall[58,59]. Although specific risk factors have not yet been identified in an evidence based manner, most authors argue that normalization of global sagittal alignment would prevent the development of junctional failure[59-61]. Junctional kyphosis is a radiographic sign which does not always produce symptoms and which shows no correlation with clinical outcomes in most studies[55,56,59,62]. As such, treatment should be reserved for those patients who are symptomatic or where there is obvious deformity. There is a single study in the literature addressing treatment of symptomatic proximal junctional kyphosis. The corrective procedures performed were Smith-Petersen osteotomies in the majority of cases, with rib osteotomies and vertebral column resection in exceptional cases[63]. Reported results were good with a minimum follow-up of two years.

NEUROLOGICAL COMPLICATIONS

The occurrence of a post-operative neurological deficit is probably the most dreaded of all spinal complications. Despite its notoriousness, the reported incidence is only 0%-2% in most reports[64]. Injury to the nervous elements can either be direct at the time surgery, such as laceration, traction or compression of an exiting nerve root, or indirect, due to disruption of blood supply or compression. Notably, injury to the peripheral nerves may occur due to improper patient positioning, with resulting nerve palsies. Direct injury can be caused by trauma from surgical instruments of from misplacement of screws and/or hooks[65]. Disruption of the blood supply usually happens during correction of spinal deformity. The use of intraoperative neurophysiological monitoring has reduced the occurrence of neurological complications, with the Stagnara wake-up test still being used in cases with increased risk of postoperative neurological deficits[66]. Compression occurs intraoperatively from cotton patties, fat grafts or dura sealing products. In these cases, deficits will manifest immediately after surgery. Compression can also be caused from a mass lesion, such as hematoma, pseudomeningocele, epidural abscess or recurrent disc herniation. The presentation of neurologic symptoms will be insidious and almost certainly never in the immediate post-operative period.

A meticulous neurologic examination as soon as the patient wakes from surgery is of critical importance to distinguish deficits that occur intraoperatively from those that develop in the early post-operative period. The significance of this baseline examination is emphasized by the fact that imaging of the post-operative spine so soon after surgery will often be of limited value. Determining the cause of the neurological lesions depends on recollection of intraoperative events by the surgeon and his team, timing of presentation of symptoms and imaging findings, if any. Depending on the cause, management varies from patiently monitoring the course and progress of any deficits to immediate surgical exploration and correction of the underlying cause.

CONCLUSION

Surgical complications in spine surgery are not uncommon. Their significance can be minor, noticeable only as mere radiographic findings, or catastrophic, presenting with pain, neurological symptoms and progressive deformity. We chose not to include adjacent segment disease (ASD) in this overview. In our view, ASD constitutes the natural progression of the disease that was originally treated with surgery or perhaps a manifestation of wrong level selection and under-treatment. Hopefully, as our understanding of spinal pathologies becomes clearer and our therapeutic arsenal more sophisticated, the rate of complications will decrease further, minimizing the risks and distress to patients.

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