Delayed C5 Palsy after Laminectomy and Fusion for Ossification of the Posterior Longitudinal Ligament

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Abstract

Ossification of the posterior longitudinal ligament (OPLL) can cause myelopathy. Laminectomy with fusion is one surgical option for the treatment of symptomatic OPLL. In this report, we present 2 illustrative cases of unilateral C5 palsy occurring in a delayed manner after posterior decompression for OPLL. Both patients were successfully treated with conservative management. incidence, potential etiology, management and outcomes for this potentially debilitating complication are reviewed.

Keywords: C5 palsy; Cervical spine; Complication; OPLL; Ossification; Posterior longitudinal ligament; Surgery

Introduction

The etiology of ossification of the posterior longitudinal ligament (OPLL) is multifactorial and involves complex genetic and environmental factors [1,2]. If left untreated, ossification can progress markedly [3] and when compression of 20-40% of the spinal cord occurs, myelopathy can develop [4]. The current mainstay treatment for symptomatic OPLL is surgical, while conservative methods are less successful [5]. A variety of surgical approaches including corpectomy, laminectomy, laminoplasty and laminectomy with fusion have been used to treat symptomatic cervical OPLL. In this article, we present 2 cases of delayed C5 palsy after multilevel cervical laminectomy and fusion. Patient management and outcomes are reported, as well as a review of the literature with regard to the incidence, etiology, management and outcomes of this potential complication in OPLL patients.

Case Illustrations

Patient 1

A 54-year-old woman with a history of diabetes presented with progressive gait abnormality, hand dysfunction and urinary incontinence. She reported multiple falls due to gait imbalance and difficulty with tasks requiring manual dexterity such as buttoning shirts. Physical examination was most notable for mild weakness of the proximal leg muscles, diffuse sensory loss in her right leg and a wide-based unsteady gait. Imaging revealed high-grade cervical stenosis attributed to OPLL. (Figure 1). OPLL (left), and sagittal reformatted CT showing ossification of posterior longitudinal ligament (right).

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weakness of hand intrinsics and unsteady gait. Cervical MRI revealed multilevel stenosis with findings consistent with OPLL (Figure 2). The patient underwent a C2-7 laminectomy in conjunction with C2-T1 instrumented fusion. Multimodality neurophysiologic monitoring was used without significant changes noted. Postoperatively, the patient had a stable neurologic examination.

On POD 3, the patient developed left deltoid weakness (MRC grade 1/5). A course of steroids was instituted. MRI was performed and showed adequate decompression (Figure 3). On POD 5, the patient developed left biceps weakness (MRC grade 2/5). The patient was discharged and underwent extensive physical therapy. At her 5-month follow-up visit, there was significant improvement in left biceps strength (MRC grade 4/5); however, her left deltoid remained weak (MRC grade 2/5). Further physical therapy was prescribed. At her 10-month follow-up visit, left deltoid function had improved to MRC grade 4/5.

Discussion

OPLL is most commonly seen in Asia and in people of Asian descent [5], which is consistent with multiple studies suggesting a genetic component [2,6,7]. OPLL commonly develops in the cervical spine and can lead to neurologic symptoms [8]. In the setting of an appropriate clinical picture, the diagnosis of OPLL can be made via MRI, preferably of the whole spine [9]. As seen in Patient 1, other areas of stenosis can occur. While surgical decompression is the mainstay treatment for OPLL, there are currently few prognostic factors that are useful in predicting complications and postoperative outcomes [10,11]. Reported surgical complications of cervical decompression for OPLL include durotomy causing cerebrospinal fluid leak [12], spinal epidural hematoma formation [13], spinal cord herniation [14], rare bilateral phrenic nerve palsy [15] and vertebral artery injury [16].

C5 palsy occurring after surgical decompression for OPLL has been reported (Table 1). Incidence ranges from 2.6% to 18.4% and onset typically is delayed, ranging from 4 hours to 7 days [5,16-19]. Unilateral or bilateral palsies are possible [5]. Given the delayed nature of the condition, an intraoperative iatrogenic injury is not typically considered causative. The most commonly cited theory for the pathogenesis of delayed C5 palsies involves spinal cord shifting and spinal nerve tethering [20-22]. However, this theory remains controversial in the current literature [23,24]. Anatomically, C5 is a relatively short spinal nerve and has a more horizontal take-off compared to other spinal nerves in the cervical region, which predisposes the C5 spinal nerve to tethering after posterior decompression [5]. Laminectomy allows the spinal cord to displace posteriorly and potentially cause a “stretch injury,” with C5 preferentially affected due to its length [25-27]. In addition, restoration of cervical lordosis can increase the tethering effect and possibly result in a higher chance of nerve root palsy [17]. Since delayed C5 palsy has also been reported after anterior decompression, other potential etiologies have been suggested.

Another theory to the pathogenesis of postoperative delayed palsies consists of brachial neuritis occurring after cervical spine surgery. Significant stress as can occur from surgery may induce reactivation of a latent infection causing brachial neuritis and resulting in a delayed cervical palsy [28]. This mechanism parallels the pathogenesis of delayed facial nerve palsies reported after craniotomies [29], which include speculation that the complication results from reactivation of a latent herpes simplex virus [30]. It has also been suggested that cauteryization of small blood vessels during surgery can lead to ischemia of nervous tissue [26] due to an inadequate blood supply. Local reperfusion has been proposed as a mechanism of additional injury leading to deterioration of spinal cord grey matter [31,32], which predisposes patients to developing postoperative palsies [33].

While there is no standard treatment for delayed C5 palsy, a conservative regimen including neurotrophin drugs, high-pressure oxygen therapy and physical therapy has been advocated [5,17]. Persistent stenosis, particularly impacting the C5 nerve root, may be an indication for further surgery, however, outcomes are unclear. A conservative approach appears to be the main treatment modality used in cases of C5 palsy and the majority of patients appear to improve significantly, as evidenced in Table 1.

In the cases presented, both patients developed a delayed unilateral C5 palsy, occurring on the sixth and third day after surgery, respectively. No adverse intraoperative events were noted. Multimodality neurophysiologic monitoring was used without any significant changes noted, suggesting that an immediate iatrogenic event did not occur. Foraminotomies, which were performed in Patient 1, did not prevent occurrence of the C5 palsy. A specific etiology in
either case was not identified and potentially could have occurred from a "tethering" phenomenon, ischemia, or brachial neuritis. Both patients were treated successfully with extensive physical therapy. Any signs of improvement, however, did not occur for at least several months and meaningful recovery was evident at 1 year for Patient 1 and 10 months for Patient 2. Affected patients should therefore be counseled that recovery may take many months.

Conclusion

Delayed C5 palsy is a potential complication after decompressive surgery for OPLL. Various etiologies have been proposed including a "tethering" phenomenon, vascular insult and brachial neuritis. Regardless of etiology, the most commonly reported treatment has been conservative, with the majority of patients reporting improvement of symptoms.

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