Postoperative Delayed Cervical Palsies: Understanding the Etiology

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Abstract

Study Design Retrospective study.

Objective This study reviews 1,768 consecutive cervical decompressions with or without instrumented fusion to identify patient-specific and procedural risk factors significantly correlated with the development of delayed cervical palsy (DCP).

Methods Baseline demographic and procedural information was collected from the electronic medical record. Particular attention was devoted to reviewing each chart for recognized risk factors of postsurgical inflammatory neuropathy: autoimmune disease, blood transfusions, diabetes, and smoking.

Results Of 1,669 patients, 56 (3.4%) developed a DCP. Although 71% of the palsies involved C5, 55% of palsies were multimyotomal and 18% were bilateral. Significant risk factors on univariate analysis included age ($p = 0.0061$, odds ratio [OR] = 1.07, 95% confidence interval [CI] 1.008 to 1.050), posterior instrumented fusion ($p < 0.0001$, OR = 3.30, 95% CI 1.920 to 5.653), prone versus semisitting/sitting position ($p = 0.0036$, OR = 3.58, 95% CI 1.451 to 11.881), number of operative levels ($p < 0.0001$, OR = 1.42, 95% CI 1.247 to 1.605), intraoperative transfusions ($p = 0.0231$, OR = 2.57, 95% CI 1.152 to 5.132), and nonspecific autoimmune disease ($p = 0.0107$, OR = 3.83, 95% CI 1.418 to 8.730). On multivariate analysis, number of operative levels ($p = 0.0053$, OR = 1.27, 95% CI 1.075 to 1.496) and nonspecific autoimmune disease ($p = 0.0416$, OR 2.95, 95% CI 1.047 to 7.092) remained significant.

Conclusions Although this study partially supports a mechanical etiology in the pathogenesis of a DCP, we also describe a notable correlation with autoimmune risk factors. Bilateral and multimyotomal involvement provides additional support that some DCPs may result from an inflammatory response and thus an underlying multifactorial etiology for this complication.

Keywords
- postoperative C5 palsy
- cervical spine
- etiology
- autoimmune
- inflammatory neuropathy

Introduction

Delayed cervical palsy (DCP) is a recognized complication of cervical spine surgery. Although unilateral C5 paresis of the deltoid and/or biceps brachia muscles in the absence of myelopathic symptoms is the most common presentation, DCPs can also manifest bilaterally involving different and/or multiple cervical myotomes.[1] Varying degrees of sensory loss and pain often accompany weakness; however, neither is a

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requirement for diagnosis. The reported incidence ranges from 0 to 30%,[2–5] depending on the study population and procedure type, with overall mean incidence 5% via meta-analysis.[4] DCPs most commonly present within the first 3 days following surgery but have been reported up to 2 months after surgery.[2,6] Fortunately, the majority resolve within 6 months, though residual deficits may persist in up to 20%.[2]

Despite identification of the predisposing factors and plausible theories, the true etiology remains unknown. The majority of the proposed theories can be categorized as either mechanical or vascular and include nerve root traction due to postoperative cord shift or change in alignment, spinal cord ischemia, and reperfusion injury.[4,5,7–10] Excessive root traction is the most commonly cited mechanical etiology, supported by differences in preoperative foraminal width, laminectomy trough width, preexisting rotation of the spinal cord, and postlaminectomy cord drift posteriorly in patients who developed C5 palsies compared with controls. Root traction is also supported by a decreased incidence of DCPs following prophylactic foraminotomies.[11–13] Autoimmune and inflammatory etiologies such as idiopathic brachial neuritis (Parsonage-Turner syndrome) and postsurgical inflammatory neuropathy (PSIN) have less frequently been cited but have certainly been described after cervical decompression and fusion.[14,15] Other variables that have been associated with an increased incidence of DCPs include male gender and the underlying pathology (ossification of the posterior longitudinal ligament > cervical spondylomyelopathy).[6] The heterogeneity of previously established risk factors and lack of an all-encompassing theory indicate that the etiology may be multifactorial. Additionally, most previous studies focused on Asian cohorts developing DCPs following laminoplasty, specifically confined to the C5 level. Thus, the incidence in patients undergoing either laminectomy alone or cervical decompression with instrumented fusion in North American populations is not well established, as is the frequency of involvement of other cervical levels.

The present study evaluated the overall incidence of DCPs in all cervical myotomes following cervical decompression with and without instrumented fusion for degenerative disease of the cervical spine. The presence of previously established risk factors for PSIN outlined by Staff et al was also assessed relative to internal controls.[15]

Materials and Methods

Selection Criteria

All aspects of this study were approved by the Institutional Review Board and in adherence with ethical standards. We retrospectively reviewed 1,768 consecutive, current procedural terminology (CPT)-coded cervical decompressions with and without instrumented fusion at Mayo Clinic Rochester between January 2008 and December 2013 for postoperative weakness within 6 weeks of the operative date. DCPs were defined as either unilateral or bilateral, new onset or worsened motor function confined to one or more cervical myotomes. Both the operative note and the immediate postoperative exam record were carefully evaluated for weakness or pain consistent with iatrogenic injury. Patients who experienced weakness directly attributable to intraoperative injury or upper extremity pain or sensory symptoms without accompanying weakness were excluded. We also excluded all tumor and trauma cases following review of all the pertinent preoperative documentation to specifically evaluate the incidence of DCPs in patients treated for degenerative disease.

Baseline Characteristics and Assessment of Risk Factors

The electronic medical record of every patient was reviewed for baseline data including age, gender, and length of follow-up. DCPs were further characterized by myotomal involvement, either unilateral or bilateral, and worse/end motor grade. Finally, the presence of suggested risk factors for PSIN were recorded as outlined by Staff et al (intraoperative transfusions, history of diabetes, comorbid cancer, concomitant history of infection, and history of smoking).[15] Several categorical variables were further analyzed in more specific subcategories including transfusion (intraoperative versus postoperative prior to discharge), comorbid cancer (active—metastatic disease or active tumor burden; remote—no evidence of disease), concomitant history of infection (active—current use of antibiotics or chronic infection; remote—acute infection within three months), and smoking (active—smoking within 3 months of operative date; remote—any other history). A history of autoimmune disease was also recorded as positive if the patient had a documented history of 1 or more of the 81 autoimmune diseases as reported in a recent comprehensive review by Hayter and Cook (►**Appendix A**).[16] Seronegative spondyloarthropathies (ankylosing spondylitis, reactive arthritis, and psoriatic arthritis) are notable diseases of the spine with a potential autoimmune origin excluded by Hayter and Cook and thus were evaluated as a separate variable. The history of autoimmune disease was further classified into one of four categories: diabetes mellitus type I, inflammatory bowel disease (Crohn disease, ulcerative colitis), rheumatoid arthritis, and other. Underlying ossification of the posterior longitudinal ligament was not specifically evaluated given its low incidence among our study cohort, a finding consistent with epidemiologic studies completed in North America.[17] Additional analysis was performed on operative differences including approach (anterior, posterior, or both) and use of instrumentation. The intraoperative somatosensory and motor evoked potentials were reviewed when available only in patients who developed a DCP. CPT coding and chart review were used in tandem to classify procedures. When both CPT coding and reviewer classification were concordant, procedures were classified into one of several nonexclusive categories: anterior fusion, anterior diskectomy and fusion, arthroplasty, corpectomy, foraminotomy, laminectomy without fusion, laminoplasty, osteotomy, posterior fusion, and revision. Cases were included in multiple categories if applicable. Procedure categories including more than 100 cases were analyzed as a categorical variable for the entire cohort, as well as examined separately for procedure specific risk factors. Postoperative

Table 1 Characteristics of delayed cervical palsies

Characteristic	Value
Temporal profile	
Median days to onset of symptoms ± SD, *n* (range)	1 (0–14)
Median days to maximal neurologic deficit, *n* ± SD (range)	2 ± 6.8 (0–38)
Myotomal involvement	
Nerve root, *n* (%) of patients with delayed cervical palsies	
C5	40 (71.4%)
C6	12 (21.4%)
C7	20 (35.7%)
C8	13 (23.2%)
T1	3 (5.6%)

Abbreviation: SD, standard deviation.

electromyograms (EMGs) within 6 months of the operative date were also evaluated for patients with persistent weakness. These studies were evaluated for bilateral involvement, multiple myotomes, and patterns consistent with a brachial plexopathy.

Statistical Analysis

All statistical analysis was performed with JMP 10 (SAS Institute Inc., Cary, North Carolina, United States) in consultation with an institutional biostatistician. For patients undergoing multiple procedures during the period, only the first event was analyzed to exclude bias introduced by multiple representations of patient-specific factors on statistical analysis. Univariate and multivariate analyses were performed using a nominal logistical regression and likelihood ratios. The backward selection method was utilized for the multivariate analysis, retaining each variable significant on univariate analysis. A p value <0.05 was the threshold for statistical significance.

Results

There were 1,669 patients who underwent 1,768 procedures during the study period, with DCPs following the initial procedure in 56 individuals (3.4%). The majority of DCPs presented in the first 72 hours following surgery (87.5%) with maximal deficit occurring within 72 hours of onset (85.7%). The C5 myotome was involved in 40 (71.4%) cases, and 31 (55.4%) cases had multiple myotomes involved. A complete analysis of temporal profile and myotomal involvement is available in ►**Table 1**. There was bilateral involvement reported in 10 (17.9%) cases. Of note, 5 (8.9%) cases of DCPs occurred at levels not operated upon. Of these first operations, there were 842 (50.4%) cervical decompressions with fusion and 827 (49.6%) cervical decompressions only. In addition to the procedure categories analyzed in ►**Table 2**, a minority of patients underwent arthroplasty ($n = 6$), laminoplasty ($n = 6$), osteotomy ($n = 1$), or revision ($n = 21$). The analysis of demographics and the risk factors for PSIN are also summarized in ►**Table 2**.

We found significant associations between the incidence of DCP with age (positive DCP: 62.2 versus negative DCP: 57.1, $p = 0.0061$), intraoperative transfusion (positive DCP: 16.1% versus negative DCP: 6.9%, $p = 0.0231$), and history of other autoimmune disease (positive DCP: 10.7% versus negative DCP: 3.0%, $p = 0.0107$). Significant procedural factors included posterior fusion (positive DCP: 48.2% versus negative DCP: 22.0%, $p < 0.0001$), sitting (positive DCP: 7.0% versus negative DCP: 21.2%, $p = 0.0037$), and number of levels (positive DCP mean: 3.52 versus negative DCP: 2.26, $p < 0.0001$). ►**Table 3** summarizes the significant variables identified on univariate and multivariate analysis as well as provides a calculated odds ratio for each associated risk factor. On multivariate analysis, the number of operative levels ($p = 0.0053$, odds ratio [OR] $= 1.27$, 95% confidence interval [CI] 1.075 to 1.496) and nonspecific autoimmune disease ($p = 0.0416$, OR 2.95, 95% CI 1.047 to 7.092) remained significant.

►**Table 4** summarizes the risk factors for specific procedure categories with a sufficient number of cases for analysis. The risk factors identified for the entire cohort were not significant for anterior diskectomy and fusion or corpectomy. Anterior fusions (number of levels, intraoperative transfusion), posterior fusions (number of levels), foraminotomies (age, sitting, number of levels, other autoimmune disease), laminectomies without fusion (author autoimmune disease), and all nonfusion procedures (sitting, other autoimmune disease) had at least one significantly correlated risk factor.

Foraminotomies were performed in 677 cases and a DCP occurred in 24 individuals (3.5%) and at one of the roots expressly decompressed in 18 cases (2.7%). Comparing those not undergoing a foraminotomy to those who did, there was no significant difference in the rate of a DCP. Chi-square analysis was done both including cases occurring at a root not decompressed with a foraminotomy (Fisher exact test $p = 0.78$) and excluding cases where a DCP occurred at a root not decompressed with a foraminotomy (Fisher exact test $p = 0.56$).

Intraoperative monitoring of somatosensory and motor evoked potentials were reviewable for 13 individuals. Only one patient exhibited any abnormality during the procedure, but the motor evoked potential instability was not consistent

Table 2 Entire cohort demographics and likelihood ratio test *p* values on univariate analysis

	Positive delayed cervical palsy	Negative delayed cervical palsy	Combined	Likelihood ratio test *p* value
Number of patients	56 (3.4%)	1,613	1,669	
Male/female, *n* (% male)	37/19 (66.1%)	999/614 (61.9%)	1,036/633 (62.1%)	0.5275
Age ± SD	62.2 ± 11.8	57.1 ± 13.7	57.3 ± 13.7	0.0061[a]
Procedure				
Anterior fusion	13 (2.6%)	483	496 (29.7%)	0.2676
With diskectomy	3 (1.7%)	171	174 (10.4%)	0.1689
Posterior fusion	27 (7.1%)	355	382 (22.8%)	<0.0001[a]
Corpectomy	5 (4.9%)	97	102 (6.1%)	0.3992
Foraminotomy	24 (3.5%)	653	677 (40.6%)	0.7228
Laminectomy (without fusion)	7 (2.6%)	265	272 (16.3%)	0.4184
Sitting	4 (1.1%)	348	352 (21.1%)	0.0036[a]
Approach				0.0554
Anterior	10 (2.0%)	482	492 (29.5%)	
Posterior	42 (3.7%)	1083	1125 (67.4%)	
Both	4 (7.7%)	48	52 (3.1%)	
Number of operative levels				<0.0001[a]
1	9 (1.2%)	721	730 (43.7%)	
2	11 (2.9%)	363	374 (22.4%)	
3	12 (5.0%)	230	242 (14.5%)	
4	11 (6.9%)	148	159 (9.5%)	
5	2 (3.0%)	64	66 (4.0%)	
6	3 (9.4%)	29	32 (1.9%)	
7+	8 (12.1%)	58	66 (4.0%)	
Previous cervical spine surgery	12 (5.0%)	226	238 (14.3%)	0.1404
Transfusion	10 (6.2%)	151	161 (9.6%)	0.0543
Intraoperative	9 (7.4%)	112	121 (7.2%)	0.0231[a]
Postoperative	4 (5.7%)	66	70 (4.2%)	0.3054
Cancer	9 (4.3%)	199	208 (12.5%)	0.4225
Active	0 (0%)	47	47 (2.8%)	0.0712
Remote	9 (5.6%)	152	161 (9.6%)	0.1255
History of smoking	22 (3.3%)	635	657 (39.4%)	0.9902
Active	8 (2.8%)	275	283 (17.0%)	0.5799
Remote	14 (3.7%)	360	374 (22.4%)	0.6404
Infection	1 (2.2%)	45	46 (2.8%)	0.6307
Active	1 (4.0%)	24	25 (1.5%)	0.8609
Remote	0 (0%)	21	21 (1.3%)	0.2297
Diabetes mellitus	10 (4.1%)	236	246 (14.7%)	0.5142
Type I	0 (0%)	23	23 (1.4%)	0.2086
Type II	10 (8.5%)	213	223 (13.4%)	0.3342
Seronegative spondylotic arthropathies	0 (0%)	14	14 (0.8%)	0.3273
History of autoimmune disease	7 (5.1%)	130	137 (8.2%)	0.2648
Rheumatoid arthritis	1 (2.3%)	42	43 (2.6%)	0.6884
Inflammatory bowel disease	0 (0%)	20	20 (1.2%)	0.2412
Diabetes mellitus (type I)	0 (0%)	23	23 (1.4%)	0.2086
Other	6 (10.9%)	49	55 (3.3%)	0.0107[a]

Abbreviation: SD, standard deviation.
[a]$p < 0.05$.

Table 3 Univariate logistic regression and multivariate logistic regression for significant risk factors for entire cohort

	Univariate analysis			Multivariate analysis		
Variable	**OR**	**95% CI**	***p* Value**	**OR**	**95% CI**	***p* Value**
Age (per year)	1.07	1.008–1.050	0.0061[a]	1.01	0.993–1.037	0.1815
History of other autoimmune disease	3.83	1.418–8.730	0.0107[a]	2.95	1.047–7.092	0.0416[a]
Intraoperative transfusion	2.57	1.152–5.132	0.0231[a]	0.85	0.346–1.890	0.6966
Number of levels (per level)	1.42	1.247–1.605	<0.0001[a]	1.27	1.075–1.496	0.0053[a]
Posterior fusion	3.30	1.920–5.653	<0.0001[a]	1.54	0.781–3.020	0.2133
Sitting	0.28	0.084–0.689	0.0036[a]	0.42	0.123–1.103	0.0816

Abbreviations: CI, confidence interval; OR, odds ratio.
[a]$p < 0.05$.

Table 4 Significant risk factors for specific procedure categories

	Anterior fusion	Anterior diskectomy and fusion	Posterior fusion	Corpectomy	Foraminotomy	Laminectomy without fusion	Nonfusion (all)	Entire cohort
n	496	174	382	102	677	272	827	1,669[b]
DCP incidence (%)	2.6	1.7	7.1	4.9	3.5	2.5	2.4	3.4%
Risk factors (*p* values)								
Age	0.2421	0.7626	0.1474	0.3994	0.0228[a]	0.4378	0.2002	0.0061[a]
Sitting	–	–	–	–	0.0009[a]	0.5206	0.0308[a]	0.0036[a]
Number of levels	0.0082[a]	0.1290	0.0188[a]	0.2089	0.0002[a]	0.4975	0.0501	<0.0001[a]
Intraoperative transfusion	0.0204[a]	0.7075	0.9700	0.1772	0.1128	0.2975	0.1514	0.0231[a]
Autoimmune other	0.4860	0.6449	0.4289	0.5218	0.0038[a]	0.0184[a]	0.0018[a]	0.0107[a]

Abbreviation: DCP, delayed cervical palsy.
[a]$p < 0.05$.
[b]Several cases are included in more than one procedure category.

with the myotome affected postoperatively. EMG results were available for 17 patients (30%) postoperatively (►**Table 5**). The incidence of bilateral (24%) and multilevel level (88%) involvement was increased when compared with clinician evaluation consistent with the DCP. Additionally, 5 (29%) were consistent with a brachial plexopathy and less indicative of a process occurring at the nerve root.

The median neurologic follow-up for our cohort is 15.6 months (range 0.1 to 65.84 months). Unfortunately, there has been no recovery of motor function in 4 (7.1%) patients. However, for those with documented improvement, the first increases in motor grade were observed a median of 22.5 days postoperatively (range, 1 to 424). At last follow-up, the majority of individuals had regained normal ($n = 27$, 48.2%) or near normal ($n = 12$, 21.4%) strength.

Table 5 Postoperative electromyography characteristics in patients developing delayed cervical palsies

Characteristic	*n* (%)
EMG within 6 mo	17 (30.4%)
Bilateral involvement	4 (23.5%)
Multilevel	15 (88.2%)
Pattern consistent with brachial plexopathy	5 (29.4%)

Abbreviation: EMG, electromyogram.

Discussion

Postoperative DCP is a known complication of cervical spine surgery with a reported incidence around 5% based on a recent meta-analysis.[4] In the current study, we demonstrated that DCP occurred in 3.4% of our cohort, and the majority of these cases involved the C5 myotome. Just greater than half of cases included multiple myotomes, and 18% were bilateral. Although abnormal transcranial electrical stimulation-induced evoked potentials are highly sensitive and specific for radiculopathy that manifests immediately upon waking from anesthesia, DCP injury does not exhibit any signs of a potentially injurious event.[18] Although nerve root irritation remains a possible consideration, our limited intraoperative monitoring results support the suggestion that these palsies

were not likely the result of intraoperative trauma or positioning. At final follow-up, 7% had stable or worsening weakness. The rate of improvement reported in our cohort is slightly better than the 80% noted in the literature,[4] though likely attributable to variability in follow-up and the definition of *improvement*.

Some authors have suggested prophylactic foraminotomies may lead to decreased rates of DCP, particularly after laminoplasty.[3,19,20] Although we were unable to determine whether a foraminotomy was prophylactic from a retrospective review of operative reports, the rate of DCP at nerve roots decompressed with foraminotomies was not statistically different from those not operated upon. In fact, there was a slight trend toward an increased prevalence of DCP when a foraminotomy was performed. Although the protective effect of prophylactic foraminotomies would be better studied prospectively, this difference may suggest that the benefit of foraminotomy noted following laminoplasty in Asian cohorts may not be applicable to North American populations undergoing laminectomy and fusion.

Interestingly, none of the risk factors for PSIN as suggested by Staff et al were correlated with the development of a DCP[15]; however, nonspecific autoimmune disease remained statistically significant on multivariate analysis ($p = 0.0416$). This finding, in addition to the clinical presence of bilateral (18%) and multilevel (55%) involvement and involvement of roots that were not at the operative levels (9%), suggests that autoimmune reactions or nonmechanical factors potentially play a role. Further consideration of the EMG results suggest that the reported incidences of bilaterality and multilevel involvement may be underestimated. However, the higher rates of bilateral and multimyotomal palsies observed in this subpopulation may be the result of more significant or prolonged disease requiring nerve conduction studies for further evaluation. The present study also identified increased age to be significantly correlated with the development of a DCP, which is consistent with a large series by Nassr et al, which reports an association with age and C5 palsy after corpectomy and laminoplasty.[2]

We also describe several procedural risk factors that are correlated with an increased rate of DCPs on univariate analysis including posterior fusions ($p < 0.0001$), prone versus sitting (0.0036), and number of operative levels ($p < 0.0001$). Recently, the development of DCPs was reported by Yamanaka et al to be higher in patients undergoing instrumented fusion after cervical laminoplasty,[21] a finding that was replicated in this study. Although the difference in cases selected for anterior and sitting approaches may explain this finding, another potential explanation is that the anterior approach and the sitting/semisitting position could offer some anatomical advantage or minimizes manipulation of the nerve roots. Other studies have suggested that the correction of cervical lordosis that is targeted following laminectomy and fusion can close the foramen and exacerbate the compression of the C5 root in cases of preexisting foraminal stenosis.[6] It may be possible to evaluate potential differences in positioning with an anatomic study, but a complete understanding of this finding may prove elusive. The number of operative levels was extremely significant on univariate analysis ($p < 0.0001$) and the only mechanical variable that remained significant on multivariate analysis (0.0053). This result suggests that the extent of the operation is the primary risk factor for this complication, and supports the theory that mechanical factors significantly contribute to the development of this complication.

Specifying risk factors by procedure categories further elucidates evidence regarding the etiology of risk factors. The prevalence of DCP across procedure categories in the present study is similar to values reported previously and suggests that the highest prevalence of DCP is observed during posterior fusion.[6] Again, the number of operative levels appears as the dominant risk factor for both anterior and posterior fusions, which may be the result of increased traction on the nerve roots from changes in alignment/cord position and higher rates of transfusion. There is also evidence from Odate et al suggesting that one can reduce the incidence of DCPs in anterior cervical decompression and fusion cases by restricting the decompression to 15 mm and avoiding asymmetric decompressions.[22] Interestingly, sitting was correlated with a lower incidence for all nonfusion cases. Due to the limited applicability of the sitting position for anterior and instrumented cases, there was a valid concern that sitting may be a proxy variable for posterior noninstrumented cases; however, the result of this study validates that sitting may be protective for this complication. The most interesting outcome of this section is the strong correlations of nonspecific autoimmune disease and age with foraminotomies. The development of a DCP seems to correlate with those undergoing direct manipulation of the nerve root in those with preexisting autoimmune disease. Although it is purely conjecture, manipulation of the nerve roots may expose immune-privileged antigens, which could further propagate an autoimmune response in an individual with heightened immunity. The bilateral and multiple myotomal presentation as well as EMG findings supporting plexopathy in some patients lend further support to this hypothesis. Further evaluation in larger cohorts may be appropriately powered to more specifically evaluate this general result.

Although there is significant evidence for a multifactorial etiology, this study identifies the number of operative levels as the most specific etiologic factor. Age and history of nonspecific autoimmune disease are identified as risk factors for this complication, which could be useful when selecting patients for surgery and counseling them as to the risks of the procedure. From a surgical perspective, the association of extent of surgery with development of DCP may lead the surgeon to be more conservative with the decompression to decrease the risk of this complication; however, this risk must be weighed against the risk of inadequate decompression resulting in continued symptoms postoperatively. Additionally, the positioning concerns may lead to specific planning considerations to minimize risk. Finally, this study is important because it represents a Western cohort of patients undergoing cervical decompression with or without fusion. Many previous studies have focused on Asian cohorts undergoing laminoplasty operations, with only C5 myotomes.

This study is limited by its retrospective design, study population, methodology, and inclusion criteria. Although verifying procedure specifics with both CPT codes and independent chart review increases our confidence in the data set, each method has its own inherent limitations that do not disappear when they are combined. The chosen study cohort also may prevent the broad applicability of this study's results, as nearly all of the cases were originally indicated for the treatment of degenerative spine disease. This cohort is only a segment of the larger cervical spine surgery population, and the current study does not sufficiently evaluate the potentially important variable of the original underlying pathology. Additionally, in an attempt to identify all atypical DCPs, it was necessary to use rather loose exclusion criteria. Neurologic deterioration resulting from a myelopathy or exacerbation of the original pathology could present similarly to a multimyotomal DCP. Each misclassification of other similar pathologies weakens the results and conclusions of the present study. There has also been increasing evidence that radiographic measurements including anteroposterior diameter, foramina diameter, and/or cord–lamina angle can be predictive of DCPs. These and other radiographic measurements should continue to be evaluated in similar cohorts to identify predicative factors that could be easily applied to clinical practice.[23]

Conclusion

The incidence of DCP is higher in patients undergoing more extensive procedures. Although a mechanical etiology is partially supported as a cause for DCP, notable correlations with autoimmune risk factors as well as bilateral and multimyotomal involvement does support the hypothesis that some DCPs may result from an autoimmune response. The present series suggests that the etiology of DCPs is multifactorial.

Disclosures
Ryan F. Planchard, none
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Appendix A Complete list of the 81 autoimmune diseases, by selected epidemiologic and genetic characteristics

Disorder (system)	Tissue autoantibody	Molecular target	Pathogenic molecular target	Females (%)	Prevalence (per 10^5)	Location
Autoimmune disseminated encephalomyelitis	Anti-myelin	MOG	Probable	43	0.4[a]	World
Autoimmune inner ear disease	Anti-cochlear			65	<0.1	World
Batten disease/neuronal ceroid lipofuscinoses	Anti-GAD	GAD65	Probable	40	0.8	Norway
Chronic inflammatory demyelinating polyneuropathy	Anti-myelin	Myelin associated glycoprotein	Probable	30	1.3	World
Encephalitis lethargica[b]	Anti-streptolysin-O				<0.1	World
	Anti-basal ganglia					
Guillain–Barré syndrome	Anti-ganglioside GM1	PMP22	Possible	43	1.3[a]	World
	Anti-PMP22					
Hashimoto encephalopathy	Anti-thyroid microsomal antibodies	TPO	Possible	90	0.7	Italy
	Anti-TPO					
Isaacs syndrome/acquired neuromyotonia[a]	Anti-VGKC	VGKC	Probable		<0.1	World
	Anti-AChR					
Miller Fisher syndrome[c]	Anti-GQ1b			43	<0.1	World
	Anti-GM1					
	Anti-GD1a					
Morvan syndrome	Anti-VGKC	VGKC	Probable	70	<0.1	World
	Anti-neuronal AChR					
Multiple sclerosis[b]	Anti-MOG	Multiple[d]	Probable	64	58.3	World
	Anti-proteolipid protein					
Myasthenia gravis	Anti-AChR	AChR	Likely	73	5.1	USA
	MuSK					
Narcolepsy	Anti-TRIB2	TRIB2	Probable	39	30.6	USA
Pediatric autoimmune neuropsychiatric disorders associated with Streptococcal infections (PANDAS)[b]	Anti-neuronal			40	<0.1	World
	Anti-GlcNAc					
	Anti-DNase B					

Appendix A (*Continued*)

Disorder (system)	Tissue autoantibody	Molecular target	Pathogenic molecular target	Females (%)	Prevalence (per 10^5)	Location
Rasmussen encephalitis	Anti-NMDA-type GluR	NMDA-type GluR	Probable		<0.1	World
Stiff-person syndrome	Anti-GAD	GAD2	Probable	>50	<0.1	World
	Anti-amphiphysin					
Vogt–Koyanagi–Harada syndrome	Anti-Ku-Mel-1	KUMEL1	Probable	65	<0.1	World
Addison disease	Anti-21-hydroxylase	CYP21A2	Probable	63	14	World
	Anti-17 α-hydroxylase					
	Anti-P450scc					
Autoimmune hypoparathyroidism	Anti-CaSR	Calcium sensing receptor	Probable	55	<0.1	World
Autoimmune hypophysitis	Anti-pituitary cytosolic protein			90	<0.1	World
Autoimmune oophoritis	Anti-OA			100	<0.1	World
	Anti-21OH					
Autoimmune orchitis	Anti-NASP	Nuclear autoantigenic sperm protein	Possible	0	<0.1	World
Autoimmune polyglandular syndrome I (APECED, autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy syndrome)	Multiple[e]			57	0.9	World
Autoimmune polyglandular syndrome II	Anti-21-hydroxylase			75	1.7	USA
	Anti-17 α-hydroxylase					
Autoimmune polyglandular syndrome III	Multiple[f]			83	<0.1	World
Diabetes mellitus, type 1	Anti-GAD	Insulin/GAD65	Probable	45	480	World[g]
	Anti-insulin					
	Anti-ICA512					
	Anti-IA-2β					
Graves disease	TSIg	TSHR	Likely	88	629	Denmark
	Anti-TBII					

(*Continued*)

Appendix A (*Continued*)

Disorder (system)	Tissue autoantibody	Molecular target	Pathogenic molecular target	Females (%)	Prevalence (per 10^5)	Location
Hashimoto autoimmune thyroiditis	Anti-TPO	Thyroperoxidase	Probable	95	791.7[h]	USA
	Anti-TG					
Immunodysregulation, polyendocrinopathy, enteropathy, X-linked				0	<0.1	World
Autoimmune hepatitis type 1	Anti-smooth muscle antibody	Asioglycoprotein receptor	Possible	78	16.9	Norway
	ANA					
	Anti-actin					
Autoimmune hepatitis type 2	Anti-LKM-1	CYP2D6	Possible	89	3	World
	Anti-P-450 IID6					
Autoimmune pancreatitis	Anti-lactoferrin	Lactoferrin	Unlikely	33	0.7	World
	Anti-amylase α 2A					
	Anti-ACA-II					
Celiac disease	Anti-TG2	Tissue transglutaminase	Probable	57	750	USA
	Anti-gliadin					
Crohn disease[b]	ASCA			41	25	World
Pernicious anemia/atrophic gastritis	Anti-H/K	H/K ATP-ase	Likely	67	150.9	USA
Primary biliary cirrhosis	Anti-mitochondrial antibodies	Multiple[i]	Probable	89	14.6	World
Primary sclerosing cholangitis	pANCA			33	8	USA
Ulcerative colitis[b]	pANCA			65	30	World
Acquired hemophilia A	Anti-FVIII	Factor VIII	Likely	50	0.8	Wales
Antiphospholipid syndrome	Anti-cardiolipin	Beta2-GPI	Probable	74	21.5	World
	Lupus anticoagulant					
	Anti-b2GPI					
Autoimmune hemolytic anemia	Anti-erythrocyte I/i	Erythrocyte I/i	Probable	64	1.6	Norway
Autoimmune lymphoproliferative syndrome	Anti-erythrocyte			50	<0.1	World
	Anti-neutrophil					

Appendix A (*Continued*)

Disorder (system)	Tissue autoantibody	Molecular target	Pathogenic molecular target	Females (%)	Prevalence (per 10^5)	Location
Autoimmune neutropenia	Anti-NA1	B2 Integrin	Possible	54	1[b]	Scotland
	Anti-NA2					
Evans syndrome	Anti-platelet			60	<0.1	USA
	Anti-erythrocyte					
Felty syndrome	Anti-G-CSF	G-CSF	Possible	75	1.7	USA
Immune thrombocytopenic purpura	Anti-GpIIb/IIIa	Glycoprotein IIb/IIIa	Probable	70	72	Denmark
	Anti-ADAMTS13					
	Anti-glycoprotein Ib-IX					
Polymyositis/dermatomyositis	Anti-Jo1	Multiple[j]	Probable	67	5.1	USA
	Anti-Mi-2					
	Anti-CADM140					
Relapsing polychondritis	Anti-collagen II	Collagen II	Probable	66	0.4[a]	USA
	Anti-collagen IV					
	Anti-collagen IX					
Rheumatoid arthritis	Rheumatoid factor	Fibrinogen, βα	Probable	75	860[h]	USA
	Anti-CCP					
	Anti-collagen II					
Still disease[b]	ANA			62	4	World
	Anti-endothelial cell antibodies					
Erythema elevatum diutinum[b]	IgA ANCA			50	< 0.1	World
Kawasaki disease[b]				40	10[a]	USA
Microscopic polyangiitis	pANCA	Myeloperoxidase	Probable	50	2	World
Polyarteritis nodosa[b]	Anti-endothelial cell antibodies			33	6.3	World
Rheumatic fever	Anti-streptolysin O	Cardiac myosin	Likely	50	250	World
	Anti-DNase B					
Takayasu arteritis[b]				85	0.3[a]	USA
Temporal arteritis[b]				85	30	USA

(*Continued*)

Appendix A (*Continued*)

Disorder (system)	Tissue autoantibody	Molecular target	Pathogenic molecular target	Females (%)	Prevalence (per 10^5)	Location
Wegener's granulomatosis	cANCA	Proteinase 3	Probable	43	3	World
Alopecia areata	Anti-hair follicle antibodies	Trichohyalin	Possible	50	150	World
Bullous pemphigoid	Anti-BP180	Bullous pemphigoid associated glycoprotein 1	Likely	50	1	World
Cicatricial pemphigoid	Anti-BP230	Laminin-332	Likely	75	0.1	World
Dermatitis herpetiformis	Anti-TGase3	Transglutaminase	Probable	36	11.2	USA
Discoid lupus erythematosus				66	7.3	World
Epidermolysis bullosa acquisita	Anti-type VII collagen Anti-plectin	Type VII collagen	Likely	58	0.2[a]	France
Linear morphea	Anti-P80 Coilin	P80 Coilin	Probable	75	1	World
Pemphigus foliaceus	Anti-Desmoglein I	Desmoglein I	Probable	50	0.1[a]	World
Pemphigus vulgaris	Anti-Desmoglein III	Desmoglein III	Probable	60	0.5[a]	World
Vitiligo	Anti-MCHR1 Anti-SOX-10	SOX10	Possible	52	400.2	World
Behçet disease[b]	Anti-oral mucous membrane			50	1.2	World
Churg–Strauss syndrome[b]	cANCA			43	0.2	World
Cogan syndrome[b]				60	<0.1	World
Calcinosis, Raynaud phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia (CREST) syndrome	Anti-centromere Anti-fibrillarin			82	5.6	USA
Essential mixed cryoglobulinemia[b]	Anti-IgG AECA			75	1	USA
Mixed connective tissue disease	Anti-U1-ribonucleoprotein	SNRNP70		80	2.7	Japan
Polyneuropathy, organomegaly, endocrinopathy, monoclonal gammopathy, and skin changes (POEMS) syndrome[b]	Anti-MAG Anti-GM1			70	< 0.1	World

Appendix A (*Continued*)

Disorder (system)	Tissue autoantibody	Molecular target	Pathogenic molecular target	Females (%)	Prevalence (per 10^5)	Location
Scleroderma	Multiple[k]	Multiple[l]	Possible	92	24	World
Sjögren syndrome	ANA	Multiple[m]	Possible	94	14.4	USA
	SSB					
	SSA					
Systemic lupus erythematosus	Multiple[n]	Multiple[o]	Possible	88	32	World
HLA-B27-associated acute anterior uveitis	Anti-S-antigen	S-antigen	Possible	34	8.7[a]	World
Sympathetic ophthalmia				50	9	World
Goodpasture disease	Anti-basement membrane	α3(IV)NC1 collagen	Likely	25	0.5	World

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[a]Incidence data (no prevalence studies identified).
[b]Autoimmune etiology is not well established.
[c]Generally regarded as a subset of Guillain-Barré syndrome.
[d]Proteolipid protein, myelin oligodendrocyte glycoprotein, myelin basic protein, neurofilament light polypeptide.
[e]Anti-candidal enolase, anti-pituitary, anti-calcium sensing receptor protein, anti-aromatic L-amino acid decarboxylase, anti-tyrosine hydroxylase.
[f]Anti-21-hydroxylase, anti-17 α-hydroxylase, anti-thyroperoxidase.
[g]World prevalence is difficult as disease displays latitude dependence.
[h]Population limited to adults (over 18 years).
[i]Seventy-four kilodalton E2, keratin, sp100, actin.
[j]Histidyl tRNA, aminoacyl tRNA synthetase, DNA-dependent nucleosome-stimulated ATPase, EXOSC10 protein, chromodomain-helicase-DNA-binding protein 4.
[k]Anti-Scl70,anti-PM/Scl, anti-RNA polymerase III, anti-centromere.
[l]Topoisomerase I, Ro, La, Ku, fibrillarin.
[m]Ro, La, golgin.
[n]Anti-dsDNA, Anti-U1A, Anti-U2B, Anti-PCNA, Anti-Smith, Anti-SSA, Anti-SSB.
[o]U2 snRNP B, cardiolipin, fibronectin, Ro, La, histone H2A H2B, vimentin.